ETS
ISSUE BINDER

WORKPLACE
SMOKING
ISSUES

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ANXIETY

TECHNICAL
AND
STATISTICAL
REPORTS

VOLUME III

1983-1984

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WORKPLACE SMOKING ISSUES

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THIS ISSUE BINDER IS INTENDED TO PROVIDE A BASIC, COMPREHENSIVE REVIEW OF THE SCIENTIFIC LITERATURE REGARDING A SPECIFIC TOPIC ON ETS AND THE HEALTH OF NONSMOKERS.

PRIMARY STUDIES AND REVIEWS HAVE BEEN HIGHLIGHTED TO IDENTIFY (1) USEFUL OR HELPFUL INFORMATION (YELLOW HIGHLIGHT) AND (2) ADVERSE RESULTS OR OPINIONS (BLUE HIGHLIGHT).

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## I. HEALTH CLAIMS

- The claim that exposure to environmental tobacco smoke (ETS) in the workplace causes disease in nonsmokers is not justified on a scientific basis.

Exposure

- Measurements taken in offices, workplaces and public places indicate that the contribution of tobacco smoke to indoor air is minimal.[1] For example, typical nicotine measurements (which are particularly revealing because nicotine is unique to tobacco smoke) range from an exposure equivalent of 1/100 to 1/1000 of one filter cigarette per hour.[2] In other words, a nonsmoker would have to spend from 100 to 1000 hours in an office, restaurant or public place in order to be exposed to the nicotine equivalent of just one cigarette.

Lung Function

- A 1980 report[3] which concluded that nonsmokers exposed to tobacco smoke at work for 20 or more years had reduced function of the small airways compared to nonsmokers not so exposed still receives considerable attention, although it was heavily criticized for questionable data acquisition and analysis.[4] In contrast, a more recent study of 1,351 German office workers reportedly found "no evidence" that everyday exposure to



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tobacco smoke in the office or at home leads to an essential reduction of lung function in healthy adults.[5]

Lung Cancer

- Nine of the published studies on spousal smoking and lung cancer examined workplace exposure to ETS and the incidence of lung cancer in nonsmokers.[6] Not one of the studies provides adequate support for an association between ETS exposure in the workplace and lung cancer.

Allergy

- One of the most widespread beliefs, especially in the workplace setting, is that some nonsmokers are "allergic" to tobacco smoke. Scientific researchers, however, have not identified specific allergens in tobacco smoke.[7] Thus, while some individuals may react to the sight or smell of tobacco smoke, this does not mean that they are experiencing an "allergic" reaction to it.



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## REFERENCES


1. Kirk, P., et al., "Environmental Tobacco Smoke in Indoor Air," Indoor and Ambient Air Quality, eds. R. Perry and P. Kirk, London, Selper Ltd.: 99-112, 1988.

Sterling, T., et al., "Environmental Tobacco Smoke and Indoor Air Quality in Modern Office Work Environment," J. of Occup. Med. 29(1): 57-62, 1987.

Proctor, C., et al., "Measurement of Environmental Tobacco Smoke in an Air-Conditioned Office Building," Present and Future of Indoor Air Quality, eds. C. Bieva et al., Amsterdam, Elsevier Science Publishers: 169-172, 1989.

Crawford, W., "Health Effects of Passive Smoking in the Workplace," Indoor and Ambient Air Quality, eds. R. Perry and P. Kirk, London, Selper Ltd.: 203-210, 1988.

Oldaker, G., et al., "Results from Surveys of Environmental Tobacco Smoke in Offices and Restaurants," Indoor Air Quality, ed. H. Kasuga, Springer-Verlag, Berlin Heidelberg: 99-104, 1990.

2. Badre, R., et al., "Pollution Atmospherique par la Fumee de Tabac (Atmospheric Pollution by Smoking)," Ann. Pharm. Fr. 36(9-10): 443-452, 1978. Translation.

Carson, J. and C. Erikson, "Results from Survey of Environmental Tobacco Smoke in Offices in Ottawa, Ontario," Environ. Technol. Letters 9: 501-508, 1988.

Hinds, W. and M. First, "Concentrations of Nicotine and Tobacco Smoke in Public Places," New Eng. J. of Med. 292(16): 844-845, 1975.

Jenkins, R., et al., "Development and Application of a Thermal Desorption-Based Method for the Determination of Nicotine in Indoor Environments," Indoor and Ambient Air Quality, eds. R. Perry and P. Kirk, London, Selper Ltd.: 557-566, 1988.

Muramatsu, M., et al., "Estimation of Personal Exposure to Ambient Nicotine in Daily Environment," Arch. Occup. Environ. Health 59: 545-550, 1987.

3. White, J. and H. Froeb, "Small-Airways Dysfunction in Nonsmokers Chronically Exposed to Tobacco Smoke," New Eng. J. of Med. 302(13): 720-723, 1980.




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4. Adlkofer, F., et al., "Small-Airways Dysfunction in Passive Smokers," New Eng. J. of Med. 303(7): 392, 1980.

Huber, G., "Small-Airways Dysfunction in Passive Smokers," New Eng. J. of Med. 303(7): 392, 1980.

Freedman, A., "Small-Airways Dysfunction in Passive Smokers," New Eng. J. of Med. 303(7): 393, 1980.

Aviado, D., "Small-Airways Dysfunction in Passive Smokers," New Eng. J. of Med. 303(7): 393, 1980.

Fountain, L.H., "White-Froeb Study Discredited by Scientists," U.S. Cong. Record, Ninety-Seventh Congress, Second Session, Washington, D.C., U.S. Government Printing Office, E5252-5254, December 16, 1982.

Lebowitz, M., "Influence of Passive Smoking on Pulmonary Function: A Survey," Preventive Medicine 13(6): 645-655, 1984.

5. Kentner, M., et al., "The Influence of Passive Smoking on Pulmonary Function - A Study of 1,351 Office Workers," Preventive Medicine 13(6): 656-669. 1984.

Kentner, M. and D. Weltle, "Passive Tobacco Smoke Inhalation and Lung Function in Adults," Indoor and Ambient Air Quality, eds. R. Perry and P. Kirk, London, Selper Ltd.: 232-241, 1988.

6. Kabat, G. and E. Wynder, "Lung Cancer in Nonsmokers," Cancer 53(5): 1214-1221, 1984.

Garfinkel, L., et al., "Involuntary Smoking and Lung Cancer: A Case-Control Study," JNCI 75(3): 463-469, 1985.

Shimizu, H., et al., "A Case-Control Study of Lung Cancer in Nonsmoking Women," Tohoku J. Exp. Med. 154: 389-97, 1988.

Koo, L., et al., "Measurements of Passive Smoking and Estimates of Lung Cancer Risk Among Non-Smoking Chinese Females," Int. J. Cancer 39: 162-169, 1987.

Wu, A., et al., "Smoking and Other Risk Factors for Lung Cancer in Women," JNCI 74(4): 747-751, 1985.

Lee, P., et al., "Relationship of Passive Smoking to Risk of Lung Cancer and Other Smoking-Associated Diseases," Brit. J. Cancer 54: 97-105, 1986.



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Svensson, C. et al., "Smoking and Passive Smoking in Relation to Lung Cancer in Women" Acta Oncologica 28: 623-629, 1989.

Wu-Williams, A., et al., "Lung Cancer Among Women in North-East China," Brit. J. of Cancer 62: 982-987, 1990.

Janerich, D. et al., "Lung Cancer and Exposure to Tobacco Smoke in the Household," New Eng. J. Med. 323(10): 632-636, 1990.

7. Lehrer, S., et al., "Tobacco Smoke Sensitivity: A Result of Allergy?," Ann Allergy 56: 1-10, 1986.



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## II. SICK BUILDING SYNDROME

Because it is visible and easily identified by its aroma, environmental tobacco smoke is often blamed for indoor air quality problems. Government and private studies of "sick buildings" in the United States, Canada and Europe report, however, that tobacco smoke may be involved in only two percent to five percent of the buildings investigated for complaints about air quality.[1] For example, from 1981 through 1987, one company studied indoor air quality in 223 different buildings, accounting for over 39 million square feet of property, and found that ETS was a significant concern in only 10, or 4%, of the buildings [Robertson, 1988]. In another SBS database, smoking was implicated as a major contributor to complaints in only 12 of 408 (<3%) of the buildings surveyed [Collett, 1989]. The National Institute for Occupational Safety and Health (NIOSH) investigated more than 200 "sick" buildings and found that tobacco smoke was the source of claimed discomfort in only 2% of the buildings investigated. Ventilation problems were associated with half the complaints; outdoor air was considered a bigger problem than ETS [Melius, 1984]. Other investigators concluded that bacterial and fungal contamination is a major source of indoor air problems [Collett, 1989; Robertson, 1988].

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- Inadequate ventilation exacerbates all indoor air quality problems. This suggests that even a total smoking ban is not likely to affect comfort problems in 95 to 98 percent of "sick buildings."[2]

- The majority of indoor air quality problems in "sick buildings" have been traced to inadequate fresh air and poor air filtration. Because the visibility of tobacco smoke may be an indicator of inadequate ventilation, the prohibition of smoking serves to mask the real reason for poor indoor air quality--lack of proper ventilation. In addition, concentrating on tobacco smoke ignores the fact that adequate ventilation should always be provided in any enclosed space, regardless of whether or not smoking is permitted.

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## REFERENCES


1. Kirkbride, J., "Sick Building Syndrome: Causes and Effects," Health and Welfare Canada, Ottawa, Canada, 1985.

   Melius, J., et al., "Indoor Air Quality -- the NIOSH Experience," Ann. Am. Conf. Gov. Ind. Hydg. 10: 3-7, 1984.

   Robertson, G., "Source, Nature and Symptomology on Indoor Air Pollutants," Indoor and Ambient Air Quality, eds. R. Perry and P. Kirk, London, Selper Ltd.: 311-319, 1988.

   Collett, C. et al., "A Database of Problem Buildings: Learning by Past Mistakes," Present and Future of Indoor Air Quality, eds. C. Bieva et al.. Amsterdam, Elsevier Science Publishers: 413-419, 1989.

   Turner, S. and P. Binnie, "An Indoor Air Quality Survey of Twenty-Six Swiss Office Buildings," Indoor Air '90, Toronto, Canada: 27-32, 1990.

2. Sterling, T. and Kleven, S., "The Epidemiology of 'Sick Public Buildings'," Indoor Air Quality: Symposium, Buenos Aires, National Academy of Sciences of Buenos Aires: 79-107, 1989.


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## III. VENTILATION

- In 1981, The American Society of Heating, Refrigeration and Air-Conditioning Engineers (ASHRAE) issued a ventilation standard for public places (ASHRAE 62-1981).[1] The Standard established two levels of ventilation, one for areas in which smoking was permitted, and another substantially lower rate for areas where smoking was prohibited. The Standard was recently revised and reissued (ASHRAE 62-1989) with one prescribed ventilation rate, regardless of whether smoking was permitted or not. The decision to reject separate ventilation rates for smoking and nonsmoking areas was influenced by two areas of research: (1) The amount of ventilation required to remove indoor contaminants produced by humans, namely carbon dioxide and body odor, is also sufficient to remove typical amounts of ETS; and (2) ventilation rates for nonsmoking areas under ASHRAE 62-1981 were found to be inadequate and permitted airborne substances to increase, even in the absence of ETS.[2]

- Operating costs for increasing ventilation to meet specifications set forth in ASHRAE 62-1989 have been estimated at 3-5% over current annual energy expenditures for office buildings. Compliance with ASHRAE Standard 62-1989 will apparently have little effect on annual energy budgets.[3]



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## REFERENCES


1. American Society of Heating, Refrigeration and Air Conditioning Engineers, ASHRAE Standard 62-1989, "Ventilation for Acceptable Indoor Air Quality," Atlanta, ASHRAE Publications, 1989.

2. Burroughs, H., "ASHRAE: Addressing the Indoor Air Quality Challenge With Energy Conscious Design," eds. R. Perry and P. Kirk, Indoor and Ambient Air Quality, London, Selper Ltd.: 530-535, 1988.

3. Eto, J., "The HVAC Costs of Increased Fresh Air Ventilation Rates in Office Buildings Part 2," Indoor Air '90, Toronto, Indoor Air '90: 53-58, 1990.


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## IV. ACCOMMODATION

- Since the claim that exposure to tobacco smoke causes disease in nonsmokers is not scientifically justified,[1] the real issue regarding the "right" to smoke-free air is whether or not smoking should be prohibited because some people consider it to be an annoyance or nuisance.

Annoyance:

- Tobacco smoke may be an annoyance or nuisance to some people, but such complaints typically arise in poorly ventilated areas. Such complaints are most frequently associated with inadequate ventilation and to indoor substances other than ETS [See Section II: "Sick-Buildings"].

- Regulating a behavior such as smoking merely because some see it as an annoyance has undesirable consequences. Numerous individual behaviors could fall into the category of "annoyances," and to demand restrictions on all those potentially "annoying" behaviors is "to call for government regulation of everything."[2]

- Such regulations also reject the real possibility that people can work things out among themselves and may place a minority



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of individuals in the position to dictate what is "right" for everyone.

- The alternative to intrusive regulation is good manners, common courtesy and cooperation between smokers and nonsmokers. This alternative preserves the delicate balance of individual rights and allows for accommodation of everyone's desires.



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## REFERENCES


1. Wu, J., "Summary and Concluding Remarks," Environmental Tobacco Smoke: Proceedings of the International Symposium at McGill University 1989, eds. D. Ecobichon and J. Wu, Lexington, Massachusetts, Lexington Books: 367-375, 1990.

Layard, M., "Environmental Tobacco Smoke and Cancer: The Epidemiologic Evidence," Environmental Tobacco Smoke: Proceedings 1989: 99-115, 1990.

Wexler, L., "Environmental Tobacco Smoke and Cardiovascular Disease: A Critique of the Epidemiological Literature and Recommendations for Future Research," Environmental Tobacco Smoke: Proceedings 1989: 139-152, 1990.

Witorsch, P., "Effects of ETS Exposure on Pulmonary Function and Respiratory Health in Adults," Environmental Tobacco Smoke: Proceedings 1989: 169-185, 1990.

Adlkofer, F., "Biological Effects After Exposure to ETS," Indoor Air Quality: Symposium, Buenos Aires, National Academy of Sciences of Buenos Aires: 61-76, 1989.

Gostomzyk, G., "Passive Smoking - Report on an International Symposium (23-25 October 1986)," Public Health 49: 212-215, 1987.

Wynder, E. and G. Kabat, "Health Care and Society: Environmental Tobacco Smoke and Lung Cancer," Present and Future of Indoor Air Quality, eds. C. Bieva et al., Amsterdam, Elsevier Science Publishers: 135-145, 1989.

2. Williams, W., "Cigarettes and Property Rights," Clearing the Air: Perspectives on Environmental Tobacco Smoke, ed. R. Tollison, Lexington, Massachusetts, D.C. Heath and Company: 39-52, 1988.




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The Separation Issue:

- The 1986 Report of the Surgeon General on ETS suggests that separation of smokers and nonsmokers is not effective in minimizing the nonsmoker's exposure to ETS. That claim was made without scientific support. Indeed, subsequent research indicates that simple separation of smokers and nonsmokers effectively minimizes ETS exposures for nonsmokers.[1-3]

- One recent study reported that the use of designated smoking areas reduced exposure to ETS by 95%.[1] Another study of a smoking-restricted office building reported that ambient nicotine in nonsmoking areas was virtually undetectable, suggesting that ETS had a negligible impact on the nonsmoking areas of the building.[2]

- Canadian researchers, in a series of reports, presented results on levels of ETS constituents in offices where smoking was regulated and unregulated. They reported no significant differences in average ETS constituent levels between nonsmoking offices that received recirculated air from designated smoking areas and nonsmoking offices that did not receive recirculated air.[3] They concluded:

> The results indicate that the provision of a designated, but not separately ventilated smoking area can effectively eliminate or



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drastically reduce most components of environmental tobacco smoke from nonsmoking offices.

- A federally-sponsored study of ETS in aircraft cabins indicates that separation of smokers and nonsmokers is an effective means for reducing exposure to ETS.[4] The authors reported that many ETS constituent concentrations in nonsmoking sections were below the limit of detection. Similar results have been reported in other studies.[5]



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## REFERENCES


1. Vaughan, W. and Hammond, K., "Impact of 'Designated Smoking Area' Policy on Nicotine Vapor and Particle Concentrations in a Modern Office Building," J. Air Waste Manage. Assoc. 40: 1012-1017, 1990.

2. Hedge, A., et al., "Building Ventilation and Smoking Policy Effects on Indoor Air Quality and Employee Comfort and Health," Indoor Air '90, Toronto, Canada, July 29-August 3: 739-744, 1990.

3. Sterling, T. and B. Mueller, "ETS in Offices and When Smoking Is Restricted to Designated but Not Separately Ventilated Areas," Indoor Air Quality, ed. H. Kasuga, Springer-Verlag, Berlin Heidelberg: 120-129, 1990.

   Sterling, T., "ETS Concentrations Under Different Conditions of Ventilation and Smoking Regulation," Indoor and Ambient Air Quality, eds. R. Perry and P. Kirk, London, Selper Ltd.: 89-98, 1988.

   Sterling, T. and Mueller, B., "Concentrations of Nicotine, RSP, CO and $CO_2$ in Nonsmoking Areas of Offices Ventilated by Air Recirculated from Smoking Designated Areas," Am. Ind. Hyg. Assoc. J. 49(9): 423-426, 1988.

   Sterling, T., et al., "Exposure to Environmental tobacco Smoke in the Non Industrial Workplace Under Different Conditions of Ventilation and Smoking Regulation," Present and Future of Indoor Air Quality, eds. C.J. Bieva, et al., Amsterdam, Elsevier Science Publishers: 111-118, 1989.

4. U.S. Department of Transportation, "Airliner Cabin Environment: Contaminant Measurements, Health Risks, and Mitigation Options: Final Report," eds. Nagda, N.L. et al., GEOMET Technologies, Inc., Report No. DOT-P-15-89-5, December 1989.

5. Oldaker, G. and Conrad, F., "Estimation of Effect of Environmental Tobacco Smoke on Air Quality Within Passenger Cabins of Commercial Aircraft," Environ. Sci. Technol. 21: 994-999, 1987.

   Malmfors, T., et al., "Air Quality in Passenger Cabins of DC-9 and MD-80 Aircraft," Environ. Technol. Letters 10: 613-628, 1989.

   O'Donnell, A., et al., "Air Quality, Ventilation, Temperature and Humidity in Aircraft," ASHRAE Journal: 42-46, 1991.




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APPENDICES

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# Smokers' Rights In the Workplace:

## An Employee Guide



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2 How should I respond if my employer says that nonsmokers have a legal right under state or federal law to insist that smoking be banned in the workplace?

3 What are my rights as a smoker?

3 Do most companies have strict smoking policies?

3 Some anti-smokers are harassing me. What can I do?

4 How can I prevent a strict anti-smoking policy or smoking ban from being enacted, if none is required by law?

5 What should I say to someone who wants to enact a strict smoking policy or smoking ban not required by law?

6 Can my union help?

6 What are my options? What types of workplace smoking policies exist?

7 If employees complain about poor air quality, is smoke necessarily the cause?

8 How can I counter claims about the negative health effects of environmental tobacco smoke?

9 How can I respond to claims that smokers are less productive than nonsmokers?

10 How can I persuade my fellow employees to speak out against workplace smoking restrictions that are not required by law?

10 Can my employer prohibit me from smoking off the job?

11 To what extent should government regulate smoking in my workplace?

12 What can I do as a citizen to fight proposed workplace smoking restrictions?

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## Overview

Job security, wages, health benefits and child care are just some of the many issues facing workers and management today. Some of these issues stir the interest and concern of workers more than others do.

Workplace smoking is one issue of current interest. In some situations, workplace smoking is strictly governed by state or local laws or regulations. In other situations, however, employers and employees have the ability to fashion their own approach to this issue. In many of these cases, a reasonable and accommodating decision is reached. However, in some instances, further discussions are needed.

To resolve workplace smoking disputes reasonably, and to prevent unfair and unnecessary restrictions, you must fully understand the facts about workplace smoking, methods for accommodating—and satisfying—both smokers and nonsmokers, and your rights and responsibilities as a smoker. You must, of course, also be aware of any laws that apply.

What follows are answers to common questions about smokers' rights in the workplace. If your employer has adopted an unfair policy, is considering adopting a smoking policy or is currently drafting one—or if you are being harassed by your employer or other employees for smoking on or off the job—this booklet is designed to help you. For further information, including information about applicable laws or regulations, contact The Tobacco Institute at 1875 I Street, Northwest, Washington, D.C. 20006.

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**How should I respond if my employer says that nonsmokers have a legal right under state or federal law to insist that smoking be banned in the workplace?**

According to state law, employers must provide a reasonably safe working environment. Anti-smoking advocates sometimes interpret this obligation as giving them a right to demand that smoking be banned in the workplace. Only once, in a lower court ruling in 1976 in New Jersey, has a court agreed with such an interpretation. This decision was limited to New Jersey, and the theory was later rejected in a case in the same state in 1983.

In the second case, an employee tried to force her employer to adopt a variety of smoking restrictions. In ruling against the employee, the court wrote

> What we are really being asked for here is to impose upon every employee . . . who wishes to smoke a regime, a form of discipline which goes well beyond the reasonable, all under the guise of catering to the very particularized needs of a supersensitive person. That is not appropriate.

The court concluded:

> [T]here simply is no warrant and no justification as a matter of civilized management of a work force to treat smokers as though they were moral lepers and to banish them to a remote isolated area of the workplace and that [w]hen one gets right down to it it is essentially what the plaintiff it seems to me is asking for in this case.

Claims that the U.S. Constitution guarantees a totally smoke-free environment also have been unanimously rejected by the courts. In that connection, a federal district court judge in Louisiana wrote in 1976 that to find in the Bill of Rights of the U.S. Constitution a right to a smoke-free environment . . .

> would be to mock the lofty purposes of such amendments and broaden their penumbral protections to unheard-of boundaries . . . The inevitable result would be that type of tyranny from which our founding fathers sought to protect the people by adopting the first ten amendments.

Nevertheless, many local governments, and some state governments as well, have enacted laws that govern smoking in the workplace. If your employer tells you that he is simply attempting to comply with applicable laws or regulations, ask him to show you the precise legal basis for his actions. Then contact your bargaining representative—or The Tobacco Institute—to determine whether the law requires your employer to act as he proposes to do.



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## What are my rights as a smoker?

Apart from any requirements established by law, your rights are determined by common standards of human behavior. For example, you are entitled to common courtesy and respect from others for your personal lifestyle and preferences—including your choice to smoke. If your employer decides to implement workplace smoking restrictions, you should insist on the right:

- To be consulted before a policy is adopted
- To be reasonably accommodated by the policy
- To have your preferences considered on an equal basis with nonsmokers
- To take any dispute or policy discussion to your union, if you are represented by one
- To be free of harassment, verbal or otherwise, and
- To be told the legal basis for the policy.

## Do most companies have strict smoking policies?

No. Very few companies completely prohibit smoking. In fact, 81 percent of companies permit smoking.

Most employers prefer to review smoking disputes on a case-by-case basis and to accommodate both smoking and nonsmoking employees. The firms that have developed policies have done so primarily to reduce smoking around sensitive equipment or food processing areas or to comply with city or state regulations.

## Some anti-smokers are harassing me. What can I do?

Arrange a private meeting to raise your concerns with those who are harassing you. Discuss your differences. Work on ways to prevent them from straining office relationships further.

If they won't cooperate or meet, say firmly that you don't want to alarm management unnecessarily with your concerns. Say that, on the contrary, you would prefer to work out the issue quietly and calmly amongst yourselves.

If that doesn't work, arrange a meeting with management to discuss the issue. Request the presence at the meeting of the person harassing you and your supervisor as a gesture of willingness to reach a solution that is balanced and fair to all parties. At the meeting, express your interest once again in reaching a solution that accommodates everyone.



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**How can I prevent a strict anti-smoking policy or smoking ban from being enacted, if none is required by law?**

Schedule a meeting to discuss the issue with those responsible for developing the policy or ban. Before the meeting, do some research:

- Find out the company's existing smoking policy. If none exists, find out if any local or state ordinances apply.
- Talk to friends at companies like your own and ask about any smoking restrictions there. Reasonable smoking policies may exist within your industry that could help your company.
- Research "successful" smoking policies that try reasonably to accommodate both smokers and nonsmokers. (A total smoking ban is not a reasonable accommodation!)
- Consider possible mechanical or structural solutions. Is your workplace properly ventilated? Would partitions reduce cigarette smoke and noise? Ask management to look into these possibilities.
- Identify informal solutions that could work in your work environment. For instance, you might try either moving individual smoking or nonsmoking employees or building physical barriers between smoking and nonsmoking employees.
- If a smoking policy is being imposed by management without employee input, ask that the workforce be consulted. Firmly but politely assert your rights.
- If you are represented by a union, discuss your concerns with your steward or other representative. Smoking policy decisions are subject to collective bargaining agreements in most cases. However, the union must speak up for its rights quickly! In addition, your union can ask management to call in an indoor air expert to analyze whether smoke is truly a problem. Most often, poor ventilation—not cigarette smoke—is at the root of poor indoor air quality.
- Recruit support from smoking and nonsmoking coworkers. The larger and more diverse your group, the more likely management will listen *and* respond to your concerns. If necessary, ask workers to sign a petition or letter of support. It's important to conduct such activities before or after work. Employers generally are more open-minded toward initiatives organized on employee rather than working time.

Now you are ready for that meeting. You've reviewed your situation and can sit down with those who are developing the policy to discuss the matter reasonably.



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**What should I say to someone who wants to enact a strict smoking policy or smoking ban not required by law?**

Arrange a meeting with those who want the restrictions. During the meeting . . .

- Don't be angry or aggressive. Rather, keep a poised and professional appearance. Stress your willingness to work together to develop a smoking policy based on common sense and courtesy.
- Express your eagerness to reach a solution based on accommodation and compromise. Accommodations often are made for others in the workplace such as allowing employees to listen to music with earphones and moving worksites to avoid annoying neighboring employees. Similar accommodations should be extended to smokers. As one New Hampshire smoker recently noted, "The smell of coffee makes me nauseous and so do some perfumes. Should we ban those things, too?"
- Display your knowledge about the workplace smoking issue. It might reverse feelings toward an all-out smoking ban. Here are some facts you can mention:
  - —There is no conclusive research that shows that smokers are less productive employees than nonsmokers. Nor do smoking employees cost their employers more.
  - —Environmental tobacco smoke usually is not the cause of the problem, but rather a symptom of a problem—poor ventilation, poor filtration and general contamination of indoor air.
  - —Regulating workplace smoking and settling office disputes are not prominent concerns of most personnel managers, and rightly so.
  - —When smoking disputes arise, most firms prefer to settle them in a fair and positive manner rather than punish individuals who smoke.
  - —Be prepared to suggest two or three alternatives to a smoking ban.
  - —Report the support in the office for an informal smoking policy that satisfies both smokers and nonsmokers.
  - —If, as a result of the meeting, you are unsuccessful in reaching an agreement, arrange to have a follow-up meeting. Suggest appointing a working group of smoking and nonsmoking managers and employees to resolve the issue. Or request the participation of representatives of labor and management.



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Source: https://www.industrydocuments.ucsf.edu/docs/zqyx0000

## Can my union help?

Yes. If you are represented by a union, take your concerns to your steward or other representative. Your union has a right to a voice in determining the rules that affect your workplace. That includes rules about smoking.

Unions can represent your interests through collective bargaining with your employer. They can help to ensure that the smoking restrictions in your office are not dictated by anti-smokers alone, but rather through joint labor-management agreement.

The AFL-CIO has stated that issues related to smoking on the job can best be worked out voluntarily in individual workplaces between labor and management in the manner that protects the interests and rights of all workers.

## What are my options? What types of workplace smoking policies exist?

Subject to applicable laws and regulations, employees' options on workplace smoking vary with the layout and circumstances of their worksites. Sometimes, an informal agreement among employees and management defining the respective rights of smokers and nonsmokers will fill the bill.

For example, management can designate an entire work area as a smoking area, giving employees the option of designating their individual work stations as smoking or nonsmoking. Employees would be encouraged to resolve their disputes among themselves. Supervisors may intervene if necessary to make sure both smoking and nonsmoking employees are satisfied.

Formal smoking policies often are more detailed. Generally, they designate where employees can and cannot smoke, for example, in conference rooms and restrooms. Because they are more detailed in what can and cannot be done and how they must be enforced, supervisors have less say in resolving employee differences and cannot be as flexible in accommodating all employees.

Since most formal smoking regulations cannot be adapted to each and every workplace, a flexible approach is often the best avenue. For instance, smoking employees can be moved closer to other smokers, away from those who do not like tobacco smoke. Or, nonsmokers can have partitions that not only stop the direct flow of smoke but also direct cool air at their desks. The end result is that both smokers and nonsmokers are accommodated.



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Source: https://www.industrydocuments.ucsf.edu/docs/zqyx0000

**If employees complain about poor air quality, is smoke necessarily the cause?**

No. Reports of sore eyes and headaches, usually high employee absentee rates and visible signs of poor ventilation, such as dirty air ducts and stale air, are symptoms of a very real problem known as "sick building syndrome."

Cigarette smoke often is mistaken as the primary cause of indoor air pollution. Tobacco smoke is visible; allergenic fungus, bacteria and invisible gases and fumes that cause symptoms such as coughing, sneezing and watery eyes are not.

In over half the buildings studied over a 15-year period for air quality complaints by the federal government's National Institute for Occupational Safety and Health (NIOSH), poor or inadequately maintained ventilating systems were determined to be the cause.

Firms specializing in analyzing indoor air quality have found evidence of incredible filth in building ventilation systems. This dirt contaminates the air employees breathe and prevents proper air flow. Thirty-eight percent of the buildings studied by one firm, ACVA Atlantic Inc., of Fairfax, VA, were found to have excessively dirty ductwork, including a "pet cemetery of dead rodents, pigeons, snakes and cockroaches."

ACVA Atlantic also found that 34 percent of the buildings they studied had no fresh air intake. To save energy costs, building managers had cut off the amount of fresh air coming into the building. The idea was to save money on heating the building during the winter and cooling it during the summer.

Thirty-one percent, moreover, had large amounts of allergenic fungi known to cause sore eyes, sore throats and other allergic symptoms similar to those sometimes blamed on environmental tobacco smoke.

One way to prevent unnecessary and unwarranted smoking restrictions is to ask your employer to run a check on the quality of air in your building. If there is a "sickness," often it can be cured by a flick of the switch on the building's air-handling system to bring more fresh air into the building. Sometimes, all the system may need is a routine cleaning.



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Source: https://www.industrydocuments.ucsf.edu/docs/zqyx0000

## How can I counter claims about the negative health effects of environmental tobacco smoke?

Despite the lack of convincing evidence, the popular misconception persists that environmental tobacco smoke (ETS) has been shown to be harmful to nonsmokers in the workplace. However, the Surgeon General has stated that the data on workplace exposure to ETS are "limited and inconclusive." No reliable scientific proof exists to support the notion that smoke poses a risk to nonsmokers in the workplace.

After reviewing the scientific literature on environmental tobacco smoke, in a review critical of ETS, the National Academy of Sciences said available scientific evidence did not show that "smoking on the job or in public places, such as restaurants, [jeopardized] the health of nonsmokers."

An international conference on indoor air quality in London in June 1988 questioned not only the quantity of evidence but also the quality of the scientific methods by which environmental tobacco smoke and poor health has been linked.

Ready for a few more facts? Research has shown that to be exposed to the nicotine equivalent of one cigarette, a nonsmoker would have to fly in the nonsmoking section of an airplane for 224 straight hours, sit in a restaurant for 17 days and nights or work in an office for 550 continuous hours.



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Source: https://www.industrydocuments.ucsf.edu/docs/zqyx0000

## How can I respond to claims that smokers are less productive than nonsmokers?

Such assertions appear unfounded, and, at best, impossible to verify. Scientific establishment of these claims, notes Marvin Kristein, a consultant to the American Health Foundation, "would require studies and data we do not now—and most likely will never—possess."

Even William Weis, the Seattle University accounting professor who first proposed the notion that smokers are less productive concedes that evidence linking employees, smoking and productivity is inconclusive. "Skeptics might argue that these numbers are as soft as the underside of a porcupine," he has written, "and that may be true."

Two-thirds of union officials and supervisors in government and industry say smoking has no significant effect on employee productivity. And, more than 90 percent believe that smoking has no significant positive or negative effect on employee productivity once employees return from smoking during their workbreaks. The same survey found that 64 percent believed a total smoking ban would negatively affect employee morale, leaving smokers alienated and feeling like second-class citizens.

Claims by smoking-restriction advocates that smokers are absent more often than nonsmokers *because* they smoke are unfounded. Experts on both sides of the issue agree that many factors are involved in absenteeism, including age, sex, family responsibilities, personal problems, job satisfaction and commuting time.



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Source: https://www.industrydocuments.ucsf.edu/docs/zqyx0000

**How can I persuade my fellow employees to speak out against workplace smoking restrictions that are not required by law?**

Approach them individually, before or after work or on breaks, to discuss the restrictions in a calm and sensible manner. The emotions that frequently surround the issue encourage misunderstanding and often distort the issue. In your discussions . . .

- Mention that inter-office differences about smoking have existed for years, and that people always have been able to settle their differences through common sense and courtesy.
- Suggest that while cigarette smoke may be an annoyance, it is difficult to regulate annoyances, much less police them. Encourage greater understanding by talking about other annoyances in your own workplace—a colleague's blaring radio, excessive talking or overwhelming cologne. Ask how they would handle complaints about these, compared to those about tobacco smoke.
- Emphasize that by asking management to intervene in office smoking disputes or to establish formal smoking restrictions, employees concede greater control to managers than is necessary—or wise.
- Finally, point out that most formal workplace smoking policies affect secretaries and clerks in open-space areas, not professionals in private offices, and therefore discriminate against the majority of women and minorities who traditionally occupy secretarial and clerical positions.

**Can my employer prohibit me from smoking *off* the job?**

Only if you let them. Allowing employers to dictate the personal lifestyle of workers both on *and* off the job is a wholly unjustified invasion of your privacy. If your employer establishes such a policy, assert your rights.



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Source: https://www.industrydocuments.ucsf.edu/docs/zqyx0000

**To what extent should government regulate smoking in my workplace?**

No conclusive scientific proof exists to support the claim that exposure to environmental tobacco smoke in public places is a health risk to nonsmokers. Still, some government officials attempt to promote a "smoke-free society" by focusing on speculation rather than scientific fact. Although the evidence on the smoking and the nonsmoker health issue is inconclusive, they feel pressured to jump aboard and, in some cases, lead the nonsmoking bandwagon.

U.S. Rep. Charlie Rose (D-N.C.) spoke out against such efforts at a recent congressional hearing to consider a proposed ban on smoking in federal offices. He said, "Let's get the science straight. It's not straight right now. . . . [We're] basing this so-called 'necessary act' on very shaky science." Rose's colleague, Rep. Harold Rogers (R-KY) rose to support him and to stress the time-tested success of common sense and courtesy.

George Cooper, a vice president of the New York Chamber of Commerce, opposed government intervention for other reasons. He questioned whether "a single specific law can deal with smoking conditions in the 190,000 business establishments in the city of New York." Cooper noted that since every business is unique, a better approach would be to address the issue at the company level.



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Source: https://www.industrydocuments.ucsf.edu/docs/zqyx0000

## What can I do as a citizen to fight proposed workplace smoking restrictions?

Letters are a relatively easy and inexpensive method to oppose smoking restrictions. They can create awareness of a pending proposal to restrict smoking and can persuade others to join your fight. In doing so, they become a very valuable and effective tool.

Smokers can write several types of letters—to the editor of your local newspaper, to your elected representatives and to your corporate management.

If you decide to write, identify yourself and explain simply and directly from the start why you are writing.

Example 1: I am very disturbed by Harry Smith's recent article (*Times*, Feb. 2) regarding smoking in public places . . .

Example 2: As an employee in Widget International's Detroit facility, I am very concerned about the proposed smoking restrictions under consideration by the company . . .

Example 3: As a small business owner, I oppose City Ordinance Number 4567, which would restrict smoking in public places . . .

Then, briefly summarize several supporting points—including any of those you have read here. Letters should be neatly written and not exceed one page. They also should include your home address and daytime phone number.

It's a good idea to send copies to others. Letters to the editor, for instance, also could be sent to your city council member, state representative or congressperson. Letters to your government representatives also can be sent to your newspaper editor or the editorial director of your local television station.

Some employers feel it necessary to establish workplace smoking policies. Some do not. For those who do, smokers are encouraged to work with their colleagues and supervisors to come up with a policy that is balanced and fair to all based on common sense and courtesy.



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If you have additional questions on the workplace smoking issue, or would like more information, write:

The Tobacco Institute
1875 I Street, Northwest
Suite 800
Washington, D.C. 20006

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# Smoking in the Workplace: Some Considerations

The Tobacco Institute
1875 I Street Northwest
Washington, DC 20006

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Today, management and workers alike are faced with a barrage of information and misinformation regarding workplace smoking issues.

Smokers and nonsmokers have lived and worked together in harmony for generations. Occasional disputes about when and where to light up have been settled individually, with common sense and courtesy. Now, there are some who would substitute laws and fines.

**Disputes about when and where to light up have been settled with common sense and courtesy**

In many cases, proponents of workplace smoking restrictions are simply trying to force their own lifestyles on others. Unsuccessful in attempts to persuade many adults to quit smoking, those calling for broad restrictions are trying to keep people from smoking by segregating or otherwise punishing them for exercising their right of free choice.

These same supporters of workplace smoking restrictions also argue that this issue is a number one priority with management. Statistics, however, suggest otherwise.

In November 1988, The Washington Legal Foundation sponsored a survey of personnel policies in Fortune 1000 companies to determine what senior human resource managers identify as the most impor-



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Source: https://www.industrydocuments.ucsf.edu/docs/zqyx0000

tant workplace issues, problems and concerns, and to understand the relative importance of various specific personnel policies. Sixty percent of senior human resource managers polled cited maintaining a competent and contented work force as their greatest challenge.

The survey also revealed that managers perceive wages, job security, and health benefits as the top priorities of employees. Only two percent cited workplace smoking as an "important human resource problem."

Nevertheless, some anti-smokers continue to press for restrictions, claiming that cigarette smoke is a health hazard to nonsmokers; others say smokers are more costly to their employers than nonsmokers. Still others claim a legal right to a smoke-free environment.

**Environmental tobacco smoke has not been shown scientifically to pose a health hazard to nonsmokers**

## Health Hazard Not Proven

A detailed review of the scientific literature on environmental tobacco smoke (ETS) yields two basic conclusions:

- First, that ETS has not been shown scientifically to pose a health hazard to nonsmokers.
- Second, as a National Academy of Sciences panel noted recently, more and better research needs to be done.

In 1985, the International Agency for Research on Cancer (IARC) reviewed the quality of the evidence in the published reports then available on the ETS/lung cancer question. The IARC was critical of ETS research reports, concluding that "each is compatible either with an increase or with an absence of risk." In other words, none established risk with any certainty.

U.S. Surgeon General C. Everett Koop acknowledged in December 1986 that more than half the studies used in his report on "involuntary smoking" found no consistent, statistically significant relationship between ETS and lung cancer in nonsmokers. In fact, a much higher percentage of the studies that have been done fail to find any such relationship.

A National Academy of Sciences (NAS) committee charged with reviewing the literature related to ETS reported late in 1986 that there is nothing in the scientific literature to support the conclusion that casual exposure to environmental tobacco smoke in public places—including the workplace—constitutes a health risk to nonsmokers.

Refutations of the premise that exposure to ETS causes adverse health effects in nonsmokers were also recorded in a February 1986 report in *Medical World News* that stated that "[s]olid scientific evidence of passive smoking's health risks to nonsmokers is as elusive as the smoke itself."

Yet another study in the July 1986 edition of the *British Journal of Cancer* concludes that environmental tobacco smoke carries no significant increase in risk of lung cancer, bronchitis or heart disease.

Serious weakness in ETS investigations cited by the Surgeon General have been noted by prominent independent scientists. The sci-

**"Should lawmakers wish to take legislative measures with regard to passive smoking, they will, for the present, not be able to base their efforts on a demonstrated health hazard from passive smoking."**





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**A National Academy of Sciences panel reported that there is nothing in the scientific literature to support the conclusion that casual exposure to ETS in the workplace constitutes a health risk to nonsmokers**

entific community continues to point up the flaws in interpretation as well as some research methodologies that have been applied. Respected German biostatistician K. Uberla, in a 1987 review of the statistical evidence published in the *International Archives of Occupational and Environmental Health*, stated: "The volume of accumulated data is conflicting and inconclusive. The observations on nonsmokers that have been made so far are compatible with either an increased risk from passive smoking or an absence of risk. Applying the criteria proposed by the International Agency for Research on Cancer there is a state of inadequate evidence."

In a guest editorial in the June 1987 issue of *American Review of Respiratory Disease*, two noted Harvard Medical School professors, expressing their opinions against active smoking and in support of the Surgeon General's 1986 Report, stated: "The health impact of exposure to [ETS]at work, except for its irritation, is largely unknown. The report is on its weakest ground scientifically here."

While both insisted that ETS exposure is harmful to the health of the nonsmoker, the NAS committee and the Surgeon General also acknowledged that there is insufficient evidence to prove claims that environmental tobacco smoke impairs respiratory function or causes heart disease or allergies in adult nonsmokers.

A number of studies have shown that exposure to ETS in normal, everyday environments is extremely low. For example, one recent study indicates that a nonsmoking employee in a typical New York City office would have to work nonstop for almost 24 days—more than 550 uninterrupted hours—to be exposed to the nicotine "equivalent" of one cigarette.

## Tobacco Smoke and Indoor Air Quality

Indoor air pollution today is becoming a major work issue in some office buildings. The universal use of air conditioning and increased reliance on controlled environments in modern office buildings has focused attention on "sick building syndrome."

Employee complaints of sore and watery eyes, abnormally high absentee rates and visible signs of poor ventilation, such as dust around ventilation ducts, all are potential signs of "sick building syndrome." Although visible cigarette smoke often is targeted as a cause of the problem, we know now that in many cases, it is a symptom, not a cause.

In fact, when it comes to poor indoor air quality, tobacco smoke appears to be among the least common sources. Reports by federal and private experts suggest that environmental tobacco smoke is related to discomfort of building occupants in just two to four percent of all cases. And even in those cases, studies indicate that improved ventilation may relieve the condition.

That means lawmakers and business managers will accomplish little or nothing even if they succeed

**Visible tobacco smoke usually is a symptom, not a cause, of "sick building syndrome"**





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in removing every last wisp of tobacco smoke from the workplace solely by prohibiting smoking. Complaints of health problems and irritations may persist.

The government's National Institute for Occupational Safety and Health (NIOSH) reports that of the 203 buildings it examined following occupant complaints, just four cases—or two percent—were attributable to cigarette smoke. An independent analysis of more than 300 major private and public buildings by ACVA Atlantic, Inc., an indoor air quality analysis firm, identified tobacco smoke as a major contributing factor to air quality complaints in only four percent—twelve buildings.

**Once the underlying problem of poor ventilation is corrected, so too is any problem with ETS**

While NIOSH identified ETS as a problem in a tiny handful of cases, inadequate ventilation was blamed in 50 percent. ACVA investigations reveal that improper attention to indoor air circulation was responsible in the majority of cases for the spread and breeding of infectious germs and allergenic dusts and spores—not to mention fiberglass particles, asbestos, chemical fumes and a host of other hazardous airborne particles undetectable to the eye and nose.

Under normal conditions with ventilation that is operating according to established building codes, tobacco smoke very quickly dissipates. In fact, this disappearing act confirms that the ventilation system in an indoor area is working properly. In those few cases where visible ETS persists, the ventilation must be suspect immediately.



Problems with cigarette smoke should be viewed as a "tip off" to the much more serious underlying problem of inadequate or improper ventilation. The good news is that once the underlying problem of poor air circulation is corrected, so too is any problem with ETS.

## Costs to Employers?

Eighty-two percent of the respondents to a 1986 survey of employers by the Bureau of National Affairs (BNA) for the American Society for Personnel Administration (ASPA) reported that the imposition of smoking restrictions had resulted in no cost savings or no savings that could be identified. Still, many proponents of workplace smoking restrictions cite studies that claim to show smokers are absent more frequently and incur higher insurance costs than nonsmokers.

**Some smoking restrictions may actually increase business costs**

But according to Marvin Kristein, an American Health Foundation economist who promotes economic arguments for workplace smoking restrictions, "we lack meaningful 'case-controlled' company comparisons of experience with smoking employees vs. nonsmoking employees vs. exsmokers and the impact on company cost." To achieve a scientific basis for such cost claims, Kristein admits, "would require studies and data we do not now—and most likely will never—possess." In fact, there is evidence that some smoking restrictions may actually increase business costs as smokers leave their desks for smoking breaks. For example, Dr. Robert



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Tollison, chairman of the Center for the Study of Public Choice at George Mason University, estimates that proposed legislation severely restricting smoking in federal buildings could cost $309.5 million per year—$867 per employee.

Smoking restriction advocates who argue that smokers are absent from work more often than nonsmokers rely on a statistical correlation that is weak at best. Experts such as UCLA economist Lewis Solmon argue that there is far too little evidence to draw such a conclusion.

**Numerous factors affect employee attendance, including job satisfaction, personal problems, family responsibility, commuting time, age and sex**

Solmon notes that numerous factors affect employee attendance patterns, including job satisfaction, commitment to employer, personal problems, family responsibility, commuting time, age and gender.

As to the claim that smokers incur higher medical costs, Solmon stresses that such claims are based on studies alleging that smokers have a higher accident rate than nonsmokers. But, he notes, since smokers are most often found among blue collar workers, they are more likely to be engaged in strenuous physical activity which increases the likelihood of exposure to accidents.

## Morale? Productivity?

Are smokers less productive than nonsmokers? Contrary to anti-smokers claim, much of the data suggest they are not.

Ninety-two percent of respondents to the 1986 BNA survey for ASPA said either that imposition of smoking restrictions had not increased worker productivity, or that they did not know whether an increase in productivity had occurred. Only four percent believed restrictions had in fact increased worker productivity. And a 1984 study released by University of Minnesota researchers found that people who smoke tended to be more productive than those who do not.

A survey of union representatives and managers in business, industry and government by Response Analysis Corporation of Princeton, N.J., found that among almost 2,000 local union officials and first-level supervisors:

- Two-thirds of survey respondents said employee smoking either has a positive effect or no effect on productivity.
- Seventy-eight percent said a smoking ban would not enable their organization to accomplish the same work with fewer employees.
- Only three percent of respondents agreed that "not hiring people simply because they smoke makes sense."
- Of the respondents who said their organizations restrict smoking, less than three percent said they did so because smoking interferes with job performance.

**A 1984 study by a University of Minnesota researcher found that smokers are *more productive* than nonsmokers**

## No Legal "Right"

Relevant case law provides virtually no support for the efforts of some anti-smokers to impose their views on employers and fellow workers. The courts have uniformly rejected arguments that a tobacco smoke-free





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environment is guaranteed by provisions of the U.S. Constitution.

In cases where employees have tried to use common law to impose smoking restrictions, the courts generally have sided with the employer, most recently in a 1985 decision in *Smith v. AT&T Technologies, Inc.* There, the court noted that it "specifically does not believe or find from the evidence that the tobacco smoke at plaintiff's former workplace was harmful or hazardous to his health" or to the health "of the other employees in that area."

**The courts have rejected arguments that a smoke-free workplace is guaranteed by the Constitution**

Accordingly, the court held that the employer had not breached any duty to its employees by refusing to segregate smokers from nonsmokers and to limit smoking to non-work areas of the building.

In Washington, D.C., in 1983, Judge William Pryor ruled that "Common law does not impose upon an employer the duty or burden to conform his workplace to the particular needs or sensitivities of an individual employee."

Smoking restriction advocates cite three decisions to support their claim of a universal right to tobacco smoke-free workplace: *Parodi v. Merit Systems Protection Board, Vickers v. Veterans Administration and Shimp v. New Jersey Bell Telephone.*

*Parodi and Vickers* involved claims by federal employees that their alleged hypersensitivity to tobacco smoke made them "disabled" or "handicapped" within the meaning of statutes applicable only to the federal government as an employer or to groups seeking federal aid. These cases have no relevance



aid. These cases have no relevance to the question of whether private employers have an obligation to provide a tobacco smoke-free environment.

The 1976 *Shimp* case, then, is the only one that has actually prohibited smoking in the workplace based on the theory that general common law can be used to compel smoking restrictions. A key determinant in *Shimp*, however, was the lack of any active defense by New Jersey Bell, which filed no answer to the complaint and submitted no affidavit in opposition to Shimp's request for a court order.

That the case has little precedential value is suggested by the court's dismissal of an identical complaint subsequently filed by Shimp's attorney before the same judge on behalf of another New Jersey Bell employee. In the second case, New Jersey Bell elected to defend itself.

In *Commonwealth of Pennsylvania v. Pennsylvania Labor Relations Board*, the court held that, for employees working under collective bargaining contracts, an employer cannot unilaterally impose smoking restrictions. Other decisions have established that, even where a broad "management rights" clause is contained in a collective bargaining agreement, such unilateral action may be subject to tests of reasonableness and equity.

***Parodi* and *Vickers* have no relevance to the question of whether private employers must provide a smoke-free work environment**

The AFL-CIO has taken a position designed to protect the rights of its smoking members, opposing discrimination against smokers and calling for voluntary smoking policies



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devised cooperatively between labor and management.

Employment policies that discriminate against smokers are contrary to public policy and may violate state and federal laws. These same discriminatory policies that penalize employees who smoke raise fundamental equal protection questions. It is difficult to understand how discrimination against smokers can be rationally justified on productivity grounds. A secretary will not make more typographical errors than a nonsmoker. Nor will a bookkeeper add and subtract less efficiently if he or she smokes.

**For employees working under collective bargaining contracts, an employer cannot unilaterally impose smoking restrictions**

Legal questions aside, however, who would want to discriminate against smokers if the primary motive in hiring is to employ the best individual for the job?

## Common Sense and Cooperation

Decisions involving smoking in the workplace are most appropriately committed to the good sense and common courtesy of smoking and nonsmoking employees. The question of when and how workers may smoke in the office is best settled by employer and employee consensus rather than by city council or state legislature.

The Tobacco Institute
1989

For more information on this and other issues write:
The Tobacco Institute
1875 I Street, Northwest
Washington, DC 20006



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# SMOKING IN THE WORKPLACE: ACCOMMODATING DIVERSITY

JOHN C. FOX
BERNADETTE M. DAVISON

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CALIFORNIA WESTERN LAW REVIEW

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# Smoking in the Workplace: Accommodating Diversity†

JOHN C. FOX*
BERNADETTE M. DAVISON**

## INTRODUCTION

Historically, individuals who smoked typically felt free to do so when and where they pleased. Indeed, smoking in public places, including places of work, was generally unrestricted. Today, however, the interests of smokers and nonsmokers often compete on the job. This is especially true in workplaces where employees work side-by-side for long periods of time. While some employees adamantly seek to preserve their "right" to smoke on the job, other nonsmoking employees are pushing for a "right" to a smoke-free work environment. And, while smoking disputes are still generally resolved informally by management, now legislatures, courts and unions have become embroiled in the controversy.

In this Article, the authors present an overview of workplace smoking issues by surveying relevant case law, analyzing state and local legislation, and addressing the prominent role of unions regarding smoking in the workplace. In addition, the authors offer practical suggestions to employers about how to accommodate the competing interests of both smokers and nonsmokers. In doing so,

† John C. Fox is a partner in, and Bernadette M. Davison is associated with, the law firm of Pillsbury, Madison & Sutro. The firm provides legal services to The Tobacco Institute, a trade association representing the interests of six American cigarette manufacturers. The views expressed herein are those of the authors and do not necessarily reflect the views of The Tobacco Institute or Pillsbury, Madison & Sutro.

* John C. Fox—J.D., George Washington University National Law Center (1976); B.A., University of California at Riverside (1973), Phi Beta Kappa, Cum Laude.

** Bernadette M. Davison—J.D., Hastings College of the Law (1986), Associate Managing Editor, *Hastings Law Journal*; B.A., University of California at Riverside (1983), Cum Laude.



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the authors hope to assist employers in resolving workplace smoking issues.

## I. BACKGROUND

Eighty-eight percent of all employers in the United States permit smoking in the workplace.[1] Of those employers that permit smoking, approximately one-half currently have no formal smoking policy.[2] Twelve percent of major employers in the United States ban smoking on the job.[3] Of the employers that ban smoking in the workplace, many appear to do so around food preparation stations or where employees work near combustible materials. For example, gasoline refineries and chemical processing companies ban smoking almost uniformly due to the severe safety considerations attendant to those workplaces. Similarly, product contamination concerns led the Campbell Soup Company to ban smoking in the workplace beginning in approximately 1896.

Approximately one-fourth of adult Americans smoke.[4] Higher incidences of smoking have been associated with lower income groups and blacks.[5] It is therefore possible to experience great variations in the percentage of smokers and nonsmokers in any particular work force. Accordingly, employers often face competing

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1. BNA, WHERE THERE'S SMOKE: PROBLEMS AND POLICIES CONCERNING SMOKING IN THE WORKPLACE: A SPECIAL REPORT 20 (2d ed. 1987) [hereinafter BNA, SPECIAL REPORT].

2. According to one survey of 1,100 employers, 63.8% have no formal smoking policy. A more recent survey of 623 employers indicated that 43% have no such policy. *See infra* note 109.

3. The vast majority of employers permit smoking somewhere on company premises. 51% percent prohibit smoking in open work areas and shared work spaces. BNA, SPECIAL REPORT, *supra* note 1, at 20.

4. According to 1987 statistics compiled by the Centers for Disease Control, 26.5% of adults in the United States smoke. *Id.* at 12.

5. PUBLIC HEALTH SERVS, U.S. DEP'T OF HEALTH AND HUMAN SERVS, THE HEALTH CONSEQUENCES OF SMOKING—CANCER AND CHRONIC LUNG DISEASE IN THE WORKPLACE: REPORT OF THE SURGEON GENERAL 48-55 (1985) [hereinafter SURGEON GENERAL'S REPORT].

A 1986 study conducted by the Centers for Disease Control found that black men smoked at a rate of 32.5% while white men smoked at a rate of 29.3%. The prevalence of smoking was only slightly higher among black women (25.1%) than white women (23.7%). *See Cigarette Smoking in the United States, 1986*, MORBITY AND MORTALITY WEEKLY REPORT 582 (Sept. 11, 1987) (distributed by the Massachusetts Medical Society).

A 1985 national health interview survey conducted by the National Center for Health Statistics also found greater incidences of smoking among blacks. *See* PUBLIC HEALTH SERVS, U.S. DEP'T OF HEALTH AND HUMAN SERVS, VITAL HEALTH STATISTICS: HEALTH PROMOTION AND DISEASE PREVENTION UNITED STATES 70 (1988).

For a collection of the results of numerous nationwide studies concerning the prevalence of smoking among blacks and whites, see Ethridge & Fox, *Toward a Civil Rights Approach to Smoking*, CURRENTS (April 1987) attachment 1 (published by the American Association for Affirmative Action).

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interests of a large group of smokers or an even larger group of nonsmokers. As a result, most employers find it prudent to accommodate, if possible, the competing interests of both smoking and nonsmoking employees. This is particularly true in this era of growing labor shortages, in which employers increasingly seek to retain happy and productive employees.[6]

While the decision to permit or limit smoking in the workplace is largely left to the discretion of employers, there are legal and practical limitations to such discretion. Accordingly, companies must proceed with care after a full evaluation of the facts and pertinent law. Further, employers are warned that the law regarding workplace smoking is still evolving.

The most significant limitations to management discretion regarding smoking in the workplace include:

(1) The common law duty to accommodate both smoking and nonsmoking employees;[7]

(2) Federal regulations, state statutes or local ordinances which limit smoking in the workplace;[8]

(3) The duty to bargain collectively with unionized employees about workplace smoking policies;[9]

(4) Management's obligation to accommodate nonsmoking employees who are found to be "medically hypersensitive" to environmental tobacco smoke (ETS) and who thus may be designated as "handicapped" pursuant to federal, state or local handicap laws;[10] and

(5) The mandates of Title VII of the 1964 Civil Rights Act (Title VII) and other discrimination laws, which prohibit employer policies which have a "disparate impact" on minority and female employees.[11]

Cases involving smoking may be based on constitutional rights, handicap discrimination acts, the Occupational Safety and Health Act (OSHA), Title VII, the National Labor Relations Act (NLRA), unemployment and workers' compensation laws, and common law theories, including wrongful discharge and negligence.

Discussed below are the various legal causes of action employ-

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6. A recent survey indicates that a majority of human resource managers polled are most concerned with issues related to recruiting and retaining good employees. Nearly 60% responded that their greatest challenge is maintaining a competent and contented work force. 2 WASHINGTON LEGAL FOUNDATION, A SURVEY OF PERSONNEL POLICIES IN THE WORKPLACE (1988).

7. *See infra* notes 51-66 and accompanying text.

8. *See infra* notes 67-91 and accompanying text.

9. *See infra* notes 92-106 and accompanying text.

10. *See infra* notes 29-42 and accompanying text.

11. *See infra* notes 43-50 and accompanying text.

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ees have brought on both sides of the issue.[12] In general, the courts have been quite hostile to the claims of nonsmokers seeking a legal right to a smoke-free work environment. That same conclusion also appears to be emerging in the new wave of "smokers' rights" cases. The consensus of the courts appears to be that, absent any legislative limitation on management's discretion, employers need to accommodate the competing interests of both smoking and nonsmoking employees. For example, management must accommodate "handicapped" employees who are found to be medically "hypersensitive" to environmental tobacco smoke.[13] On the other hand, unionized workers may have a right to smoke unless and until management has bargained in good faith to limit smoking in the workplace.[14] At the same time, management must comply with any state statutes or local ordinances which may address smoking in the workplace.[15]

Inevitably, employers find themselves positioned between the competing interests of smokers and nonsmokers alike. In this situation, employers are well-advised to promptly and effectively accommodate these competing interests so as to avoid disruptive battles between employees. In some situations, employers may consider voluntarily formulating a reasonable smoking policy to assist them toward this end.

## II. JUDICIAL RESPONSE TO WORKPLACE SMOKING

Workplace smoking issues have been litigated in the courts in a variety of contexts. Beginning in 1976, nonsmokers filed the first of a series of "test cases" seeking the legal right to a smoke-free

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12. This Article does not address health or product liability litigation issues concerning tobacco use. For a discussion of the scientific evidence relating to environmental tobacco smoke, *see generally* NATIONAL ACADEMY OF SCIENCES, ENVIRONMENTAL TOBACCO SMOKE: MEASURING EXPOSURES AND ASSESSING HEALTH EFFECTS (1986) [hereinafter NAS REPORT]; SURGEON GENERAL'S REPORT ON ENVIRONMENTAL TOBACCO SMOKE—THE HEALTH CONSEQUENCES OF INVOLUNTARY SMOKING (1986) [hereinafter SURGEON GENERAL'S REPORT]; 38 WORLD HEALTH ORGANIZATION, INTERNATIONAL AGENCY FOR RESEARCH ON CANCER, IARC MONOGRAPH ON THE EVALUATION OF THE CARCINOGENIC RISK OF CHEMICALS TO HUMANS: TOBACCO SMOKING 308 (1986) [hereinafter IARC MONOGRAPH]; and PROCEEDINGS OF INDOOR AND AMBIENT AIR QUALITY CONFERENCE, *Imperial College* (London, England, June 13-15, 1988) [hereinafter PROCEEDINGS].

*See also* Comment, *Judicial and Legislative Control of the Tobacco Industry: Toward a Smoke-Free Society?*, 56 U. CIN. L. REV. 317 (1987); and Crist & Majoras, *The "New" Wave In Smoking and Health Litigation—Is Anything Really So New?*, 54 TENN. L. REV 551 (1987).

13. *See* Parodi v. Merit Sys. Protection Bd., 702 F.2d 743, 749-51 (9th Cir. 1982); *see also infra* notes 37 & 38 and accompanying text.

14. *See infra* notes 92-106 and accompanying text.

15. *See infra* notes 67-86 and accompanying text.

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work environment.[16] With a few limited exceptions, the courts have been hostile to the claims of healthy nonsmokers seeking a legal right to a smoke-free work environment. Instead, the general response of the courts has been that this is an issue best left to management discretion or the legislative process.[17]

### *A. Constitutional Claims*

The courts have summarily rejected the notion that employees or members of the public have a constitutional right to an environment free of tobacco smoke. In the leading decision, *Gasper v. Louisiana Stadium and Exposition District*,[18] a group of nonsmokers sought to prohibit smoking during sports and other public events at the Louisiana Superdome. The plaintiffs claimed that their exposure to tobacco smoke in the Superdome infringed upon their rights guaranteed by the U.S. Constitution. Specifically, the plaintiffs in *Gasper* alleged that exposure to tobacco smoke at the Superdome infringed upon their first amendment right to receive ideas; deprived them of life, liberty and property without due process in violation of the fifth and fourteenth amendments; and breached their fundamental privacy rights guaranteed by the ninth amendment. The court rejected each of the plaintiffs' constitutional arguments, and stated that to hold that the Constitution prohibits smoking would be to create an unprecedented avenue "through which an individual could attempt to regulate the social habits of his neighbor."[19]

Likewise, in *Kensell v. Oklahoma*,[20] a public employee's constitutional challenge to workplace smoking was also rejected. In this case, the court unequivocally stated that "the United States Constitution does not empower the federal judiciary . . . to impose nosmoking rules in the plaintiff's workplace."[21] In sum, every court faced with the issue has concluded that employees have no constitutionally protected right to a smoke-free work environment.[22]

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16. *See* Shimp v. New Jersey Bell Tele., 145 N.J. Super. 516, 368 A.2d 408 (1976).

17. *See, e.g.*, Federal Employees for Nonsmokers' Rights v. United States, 446 F. Supp. 181, 185 (D.D.C. 1978), *aff'd*, 598 F.2d 310 (D.C. Cir. 1979), *cert. denied*, 444 U.S. 926; Gasper v. Louisiana Stadium & Exposition Dist., 418 F. Supp. 716, 722 (E.D. La. 1976); McCarthy v. Social and Health Servs., 110 Wash. 2d 812, 826, 759 P.2d 351, 358 (1988).

18. 418 F. Supp. 716 (E.D. La. 1976), *aff'd*, 577 F.2d 897 (5th Cir. 1978), *cert. denied*, 439 U.S. 1073 (1979).

19. *Id.* at 721.

20. 716 F.2d 1350 (10th Cir. 1983).

21. *Id.* at 1351.

22. *See also* Federal Employees for Nonsmokers' Rights v. United States, 446 F. Supp. 181 (D.D.C. 1978), *aff'd*, 598 F.2d 310 (D.C. Cir.), *cert. denied*, 444 U.S. 926 (1979) (smoking in federal buildings does not violate Constitution); GASP v. Mecklenburg

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Similarly, the corollary to the nonsmokers' challenge, i.e., the smoker's asserted constitutional "right" to smoke, has also been rejected. In *Rossie v. Wisconsin Department of Revenue*,[23] the state intermediate appellate court found that a Wisconsin statute prohibiting smoking in all but certain designated areas of a state-controlled building did not violate the fourteenth amendment equal protection clause.

A prohibition against off-duty smoking, a restriction thus far imposed almost uniquely upon some police, fire and other public safety officers, has also withstood constitutional challenge.[24] In *Grusendorf v. Oklahoma City*[25] the court upheld a fire department's ban on *off-duty* smoking applicable to fire fighter trainees. The *Grusendorf* court agreed with the plaintiff that the smoking ban infringed upon liberty and privacy rights, stating:

> It can hardly be disputed that the Oklahoma City Fire Department's nonsmoking regulation infringes upon the liberty and privacy of fire fighter trainees. The regulation reaches well beyond the work place and well beyond the hours for which they receive pay. It burdens them after their shift has ended, restricts them on weekends and vacations, in their automobiles and backyards and even, with the doors closed and the shades drawn, in the private sanctuary of their own homes.[26]

Despite this finding, the court upheld the smoking ban because, under the circumstances, the city satisfied its burden of proving that the ban bore a rational relationship to the promotion of the health and safety of the fire fighters. Nevertheless, employer restrictions against off-duty behavior give rise to serious privacy

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County, 42 N.C. App. 225, 256 S.E.2d 477 (1979) (smoking in county buildings and facilities not unconstitutional).

23. 133 Wis. 2d 341, 395 N.W.2d 801 (Wis. Ct. App. 1986), *rev. denied*, 134 Wis. 2d 457, 401 N.W.2d 10 (1987).

24. Fire fighters in many states are protected by "heart and lung" statutes which create a presumption that any cardiovascular or respiratory conditions suffered are work-related. These statutes are often used to justify off-duty smoking bans. *See generally* Rothstein, *Refusing to Employ Smokers: Good Public Health or Bad Public Policy*, 62 NOTRE DAME L. REV. 940, 952-53 (1987).

Most smoking bans have been promulgated at the local level. Approximately 32 localities currently discriminate against smokers when hiring fire fighters, police officers and other public safety employees. Massachusetts is currently the only state to ban smoking by newly hired recruits.

The most stringent hiring policies call for refusal to hire smokers, require signatures of agreement not to smoke, call for possible termination upon violation, and have, in some instances, been written into collective bargaining agreements. Several localities have expressed a general preference for nonsmoking employees if a choice must be made between a smoker and a nonsmoker. At least one jurisdiction requires mandatory attendance at "health seminars," which include compulsory exercise and nonsmoking educational programs.

25. 816 F.2d 539 (10th Cir. 1987).

26. *Id.* at 541.

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concerns and are likely to be challenged in the future.[27] Moreover, where the smoking restrictions apply only to part of the work force, such as new hires, they are likely to be challenged on equal protection grounds.[28] These issues have yet to be fully litigated in the courts.

### *B. Statutory Claims*

Nonsmokers have relied upon numerous statutes attempting to limit smoking in the workplace or, alternatively, to obtain financial benefits if they believe they cannot continue working in the presence of tobacco smoke. These statutory bases include federal and state handicap laws, disability statutes and workers' compensation laws.[29]

Despite these attempts, the courts have generally refused to restrict workplace smoking. Some courts, however, have held that employees claiming severe adverse reactions to tobacco smoke are "handicapped" or "disabled" or may be able to recover workers' compensation benefits.

In *Vickers v. Veterans Administration*,[30] the court found that an employee was "handicapped" within the meaning of section 504 of the Federal Rehabilitation Act of 1973[31] when it found him to be "hypersensitive"[32] to tobacco smoke and physically una-

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27. *See* Max, *The Company Is Watching You Everywhere*, N.Y. Times, Feb. 15, 1987, § 4, at 21, col. 2; Leap, *When Can You Fire for Off-duty Conduct?*, 66 Harv. Bus. Rev., 28-30, Jan.-Feb. 1988.

28. In *Grusendorf*, the court specifically questioned whether the smoking ban, which applied only to fire fighter *trainees*, could withstand an attack on equal protection grounds. The court refused, however, to consider this issue because it was not raised by the parties. 816 F.2d at 543.

29. The Occupational Safety and Health Act (OSHA), 29 U.S.C. §§ 651-678 (1982) currently provides no remedy for nonsmokers seeking a smoke-free work environment. Tobacco smoke is not listed by OSHA as a "toxic and hazardous substance." *See* 29 C.F.R. § 1910.1000-1910.1500 (1988). Indeed, in 1987 OSHA denied a citizen petition requesting OSHA to classify tobacco smoke as a potential occupational carcinogen and develop a standard for "tobacco smoke." BNA, Daily Report for Executives at A-24 (Mar. 2, 1987). In addition, OSHA administrators have consistently refused to accept complaints based solely on workplace smoking and, thus, apparently do not perceive that environmental tobacco smoke constitutes a violation of an employer's statutory duty to provide a "healthful" working environment. Moreover, OSHA provides no private right of action for employees who seek to restrict workplace smoking. *See* Federal Employees for Nonsmokers Rights v. United States, 446 F. Supp. 181 (D.D.C. 1978), *aff'd*, 598 F.2d 310, *cert. denied*, 444 U.S. 926 (1979); Barrera v. E. I. du Pont de Nemours, 653 F.2d 915, 920 (5th Cir. 1981).

30. 549 F. Supp. 85 (W.D. Wash. 1982).

31. 29 U.S.C. §§ 701-796i (1982). The Rehabilitation Act imposes affirmative action and nondiscrimination obligations upon a limited group of employers: federal agencies, federal contractors, and recipients of federal assistance.

32. The *Vickers* court used the terms "hypersensitivity" and "unusually sensitive" interchangeably. *See* 549 F. Supp. at 87. In medical terms, "hypersensitivity" is defined as "a state of altered reactivity in which the body reacts with an exaggerated response to a

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ble to perform his job in the presence of environmental tobacco smoke.[33] In *GASP v. Mecklenburg County*,[34] however, the court rejected similar claims, cautioning that the term "handicap" was *not* intended to include all persons who claim to suffer from a pulmonary problem, however minor, or those who are simply irritated by tobacco smoke.

In the workers' compensation context, a California court has held that a nurse who left her job because of "allergic" reactions to tobacco smoke was eligible for unemployment compensation until she could find alternative employment in a smoke-free environment.[35] A Louisiana court has denied unemployment benefits under similar circumstances because it found that the employee's preexisting allergy, which was not aggravated by her employment, did not constitute "good cause" for her resignation.[36]

In *Parodi v. Merit Systems Protection Board*,[37] a federal employee who claimed to be hypersensitive to tobacco smoke was found to be "disabled." Nevertheless, the *Parodi* court found that the employee would not be entitled to disability benefits if the employer offered her a reasonable accommodation by transfer to a comparable job in a smoke-free work area.[38]

While each case is based on its own set of facts, these cases seem to indicate that currently only those found to have the most

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foreign agent." DORLAND'S ILLUSTRATED MEDICAL DICTIONARY 635 (26th ed. 1981).

This term must not be confused with "allergy" or "allergic reaction." To date, no specific antigens have been identified in *tobacco smoke*, and when individuals claim to be "allergic" to smoke, at best, they can be said to suffer from non-specific responses to smoke exposure. *See* Lehrer, *Tobacco Smoke Sensitivity: A Result of Allergy?*, ANNALS OF ALLERGY 56, May 1986, at 1-10.

33. The *Vickers* court did not award any injunctive or monetary relief, however, because it found that (1) the employer did not discriminate against plaintiff by reason of his handicap; and (2) the employer made reasonable efforts to accommodate plaintiff. 549 F. Supp. at 87-89. For a thorough analysis of the *Vickers* decision see Comment, *Limited Relief for Federal Employees Hypersensitive to Tobacco Smoke: Federal Employer's Who'd Rather Fight May Have to Switch*, 59 WASH. L. REV. 305, 312-22 (1984). *See also* Department of Fair Employment and Hous. v. Fresno County, FEHC Dec. No. 81-82 (CS-0009 ph) (1984) (employee allergic to tobacco smoke found to be "handicapped" under California Fair Employment and Housing Act).

34. 42 N.C. App. 225, 256 S.E.2d 477 (1979).

35. Alexander v. California Unemployment Ins. Appeals Bd., 104 Cal. App. 3d 97, 163 Cal. Rptr. 411 (1980). *See also* McCrocklin v. Employment Dev. Dept., 156 Cal. App. 3d 1067, 205 Cal. Rptr. 156 (1984) (employee's "good-faith fear" that smoke-filled room was harmful to his health found "reasonable" and employee entitled to unemployment benefits).

36. Billman v. Sumrall, 464 So. 2d 382 (La. App. 1985). *But see* Lapham v. Pennsylvania Unemployment Compensation Bd. of Review, 103 Pa. Commw. 144, 519 A.2d 1101 (1987) (bronchitis sufferer entitled to collect unemployment benefits where proffered physical relocation was deemed not a "reasonable accommodation"); *McCrocklin*, 156 Cal. App. 3d 1067, 205 Cal. Rptr. 156.

37. 690 F.2d 731 (9th Cir. 1982), *as amended*, 702 F.2d 743 (9th Cir. 1982).

38. For a thorough analysis of *Parodi*, see Comment, *supra* note 33, at 308-22.

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severe reactions to tobacco smoke will be considered "handicapped" or "disabled."[39] On the other hand, an employee who is merely "irritated" or "annoyed" is not typically found to be "medically hypersensitive."[40] Private employers subject to state handicapped statutes, or federal contractors covered by section 503 of the Rehabilitation Act of 1973, may accordingly also have a duty to reasonably accommodate employees found to be hypersensitive where such an accommodation would not pose an "undue hardship" on the employer or the rest of its work force. Yet, as the court recognized in *Vickers*, "the desires of those employees who wish to smoke cannot be disregarded."[41]

An employer's offer to transfer the "handicapped" individual to a comparable position in a smoke-free work area would appear to be sufficient accommodation.[42] But when a medically hypersensitive employee is unable to continue working, he or she may be entitled to disability or workers' compensation benefits. Despite this fact, employers have great latitude to accommodate the competing interests of smoking and nonsmoking employees because the foregoing cases do *not* impose any affirmative obligation upon employers to restrict workplace smoking.

### C. *Discrimination Claims*

There is also a possibility that employers' policies or practices limiting smoking in the workplace may trigger "disparate impact" discrimination claims.[43] Because a greater percentage of blacks in the United States smoke than whites, outright hiring bans or other policies which unduly restrict smoking in the workplace may disproportionately affect black employees.[44] Thus, these actions could

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39. *See, e.g.*, Vickers v. Veterans Admin., 549 F. Supp. 85, 87 (W.D. Wash. 1982).

40. *See, e.g.*, GASP v. Mecklenburg County, 42 N.C. App. 225, 256 S.E.2d 477 (1979); Gordon v. Raven Sys. & Research, 462 A.2d 10, 15 (D.C. App. 1983).

41. *Vickers*, 549 F. Supp. at 89.

42. *See Parodi*, 702 F.2d at 749-51.

43. The Civil Rights Act of 1964, 42 U.S.C. § 2000e-2000e-17 (1982) prohibits employer policies which, while facially neutral, "operate to 'freeze' the status quo of prior discriminatory employment practices." Griggs v. Duke Power Co., 401 U.S. 424 (1971). In other words, Title VII prohibits otherwise neutral employment practices which disproportionately affect protected groups.

Employer smoking restrictions may also be challenged under Title VII if individuals are subjected to "disparate treatment" because of their race, sex, religion or national origin. In such cases, the plaintiffs must prove that the employer *intended* to treat them differently on account of their protected status. *See* McDonnell Douglas Corp. v. Green, 411 U.S. 792 (1973). *But see* Moore v. Inmont Corp., 608 F. Supp. 919 (W.D.N.C. 1985) (Title VII claim rejected where black employee discharged for violating smoking policy related to safety standards that was applied equally to all employees).

44. The results of smoking prevalence studies vary depending on the year and sample size. However, the studies uniformly report a greater incidence of smoking among blacks than whites. For example, in 1980, 47.7% of black males smoked, compared with 40.2%

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be challenged under state or federal discrimination laws.[45] Once an employee shows that an employer's otherwise neutral smoking policy or practice has a statistically significant disproportionate impact on blacks, the employee has made out a prima facie case of discrimination under Title VII.[46] In order to defend, the employer must then successfully demonstrate that the policy or practice is justified by a "business necessity."[47] Some smoking restrictions, such as those prohibiting smoking near hazardous or flammable materials, may constitute a "business necessity."[48] Unless required by statute or ordinance, however, it is unlikely that courts would find that the preferences of co-employees or customers rise to the level of a business necessity.[49] Even so, the court could still find a Title VII violation if the employee proves that there are other alternatives which accomplish the same business purpose, yet have less impact on blacks.[50]

Accordingly, employers need to examine carefully current or proposed workplace smoking restrictions to ensure that they do not discriminate against protected groups. If they do, the employer must be prepared to establish that the smoking policy adopted is justified by legitimate business purposes and is the least drastic means of accomplishing the employer's goals.

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of white males. SURGEON GENERAL'S REPORT, *supra* note 5, at 49. *See also supra* note 5.

45. For a comprehensive discussion of smoking and civil rights issues, see Ethridge & Fox, *supra* note 5.

46. A prima facie case is sufficient to prove a Title VII violation, unless contradicted or overcome by other evidence. McDonnell Douglas Corp. v. Green, 411 U.S. 792 (1973).

47. To prove a business necessity, an employer may show that the policy or practice has a "manifest relationship" to performance of the job in question (i.e., that it is a "job-related criterion"). Alternatively, the employer may seek to prove that the policy or practice in question is necessary to the safe and efficient operation of the business. "[A] discriminatory employment practice must be shown to be necessary to safe and efficient job performance to survive a Title VII challenge." Dothard v. Rawlinson, 433 U.S. 321, 332 n.14 (1977).

48. *See, e.g.*, Moore v. Inmont Corp., 608 F. Supp. 919, 927 (W.D.N.C. 1985).

49. *See* Rucker v. Higher Educ. Aids Bd., 669 F.2d 1179, 1181 (7th Cir. 1982) (employer is forbidden by Title VII to refuse to hire someone on racial grounds because his customers or clientele do not like his race); Diaz v. Pan Am. World Airways, 442 F.2d 385 (5th Cir.), *cert. denied*, 404 U.S. 940 (1971) (Pan Am violated Title VII when it banned employment of male flight attendants despite passenger preferences for female flight attendants); Bing v. Roadway Enter., 444 F.2d 687 (5th Cir. 1971) (invalidating a motor freight company's rule that an employee who desired to transfer to another job must resign his present position and thereby forfeit accrued employment rights. Finding the rule to have an adverse impact on blacks, the court rejected the company's argument that the rule was "necessitated" by the prospect of employee unhappiness with the demise of the rule). *Accord* Jones v. Lee Way Motor Freight, 431 F.2d 245 (10th Cir. 1970), *cert. denied*, 401 U.S. 954 (1971).

50. *See* Dothard v. Rawlinson, 433 U.S. 321 (1977). If an employer meets the burden of showing that its tests or selection devices are job-related, the burden then shifts to the complaining party to show that other less discriminatory selection devices would also serve the employer's legitimate interests. *See also* Albemarle Paper Co. v. Moody, 422 U.S. 405, 425 (1975).

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### D. Common Law Claims

An employer's general common law duty to provide a reasonably safe working environment for its employees has been codified by federal and state occupational safety and health (OSHA) laws.[51] In an attempt to restrict workplace smoking, employees have filed several lawsuits claiming that such smoking violates this general common law duty. However, only one lower court decision, in New Jersey, *Shimp v. New Jersey Bell Telephone*,[52] has found an employer permitting smoking in the workplace to have violated this duty. The remainder of the courts confronted with this issue have declined to find the duty violated and have refused to restrict workplace smoking. Thus, there is currently little, if any, authority for imposing a common law obligation upon employers to restrict smoking in the workplace.

In *Shimp*, a secretary who claimed to suffer from a severe "allergic" reaction to tobacco smoke sought an injunction to prevent other employees from smoking in her work area. Plaintiff submitted medical opinions in support of her request for an injunction. The employer, on the other hand, failed to put forth any evidence to refute the plaintiff's claims. Not surprisingly, the New Jersey Superior Court found that the employer had a common law duty to provide safe working conditions. Accordingly, it directed the employer to restrict smoking to the lunchroom.[53]

A key determinant of the outcome of *Shimp* was a lack of any active defense by New Jersey Bell, which filed no answer or affidavits in opposition to the plaintiff's request for an injunction. It is quite possible that the result in *Shimp* might have been different had it not been uncontested and had New Jersey Bell, instead, presented a true "case and controversy." Significantly, an identical complaint subsequently filed by Ms. Shimp's attorney before the same judge on behalf of another New Jersey Bell employee was summarily dismissed.[54] The only difference between the two cases is that the employer elected to defend itself in the later case.

Also, seven years later in *Smith v. Blue Cross & Blue Shield*,[55]

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51. *See* 29 U.S.C. §§ 651-678 (1982) (Federal OSHA). Section 654(a) sets forth the so-called "general duty" clause, which requires that an employer "shall furnish to each of his employees employment and a place of employment which are free from recognized hazards that are causing or likely to cause death or serious physical harm to his employees." *See also supra* note 29.

*See also* CAL. LABOR CODE §§ 6300-6711 (Deering Supp. 1988) (Cal-OSHA). Section 6400 provides that "[e]very employer shall furnish employment and a place of employment which are safe and healthful for the employees therein."

52. 145 N.J. Super. 516, 368 A.2d 408 (1976).

53. *Id.* at 531, 368 A.2d at 416.

54. Mitchell v. New Jersey Bell Tel. Co., No. C-4159-76 (N.J. Super. Ct. Ch. Div.)

55. No. C-3617-81E, 16-17 (N.J. Super. Ct., Aug. 18, 1983).

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the New Jersey Superior Court *rejected* and dismissed a similar claim by a nonsmoking employee who also claimed to be hypersensitive to cigarette smoke. The court held that the safety of the workplace was to be judged by reference to the "typical" employee, *not* the hypersensitive employee.[56] Moreover, the court limited the holding in *Shimp*, stating:

> Insofar as the *Shimp* case is read by some as requiring an employer to institute Draconian measures to smoking employees I think it has to be viewed somewhat skeptically and cautiously. I myself have no problem at all with the basic concept of *Shimp*, that a safe workplace is required, but I must say it seems to me that some of the prohibitions contained in the *Shimp* case are too sweeping and go well beyond what is necessary to ensure a safe working place.[57]

The court therefore concluded that the nonsmoking "supersensitive" employee had no right to a smoke-free environment.

A District of Columbia court reached the same conclusion in *Gordon v. Raven Systems & Research, Inc.*[58] In *Gordon*, the employer terminated an employee after she refused to work in an area containing some tobacco smoke. The employee subsequently filed a lawsuit contending that the employer was negligent in not providing her with a smoke-free workplace. The court dismissed the employee's claim, because the plaintiff had presented no evidence to support her allegations that tobacco smoke was harmful to employees. Significantly, too, the District of Columbia court held that the employer had no duty to conform the workplace to the particular needs or sensitivities of an individual employee.[59]

Likewise, the trial court in *Smith v. AT&T Technologies*[60] also rejected an employee's claim that the employer breached its common law duty to maintain a safe working environment. This conclusion was based on the court's finding that "the tobacco smoke in plaintiff's former work area was [not] hazardous to the health of plaintiff or the health of the other employees in that area."[61]

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56. *Id.* at 15. ("It simply is not right in terms of the way in which human beings have to relate to one another, that because someone is as sensitive as that all the rest of the world has to go through a tightly-controlled regimen of smoking discipline"). *Id.* at 13. ("[Smokers] are after all human beings with needs and feelings like everyone else, and there simply is no matter of civilized management of a work force to treat smokers as though they were moral lepers and to banish them to a remote isolated area of the workplace").

57. *Id.* at 8.

58. 462 A.2d 10 (D.C. App. 1983).

59. *Id.* at 15.

60. No. 4446121 (St. Louis Cty. Cir. Ct., Apr. 23, 1985). This case was on remand from an earlier decision, Smith v. Western Elec. Co., 643 S.W.2d 10 (Mo. App. 1982), which recognized that employers have a common law duty to provide a safe workplace.

61. *Smith*, No. 4446121, at 3 (St. Louis Cty. Cir. Ct., Apr. 23, 1985).

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The court added that the employer was not required to provide a "comfortable" workplace.[63]

While there is thus far only a single ruling to support the allegation that environmental tobacco smoke causes harm to nonsmokers, employers nevertheless must be careful not to retaliate against employees who protest corporate policies permitting smoking. In *Hentzel v. Singer Co.*,[64] a California court held that an employee could state a common law retaliatory dismissal claim after being terminated for protesting hazardous working conditions. The court did so without addressing whether the alleged hazard (environmental tobacco smoke) was, in fact, hazardous.

Overall, the courts have been reluctant to find any common law basis for restricting workplace smoking in the absence of sufficient proof that environmental tobacco smoke causes significant medical harm to nonsmokers.[64] With the exception of the now dated and criticized 1976 *Shimp* decision, the courts have declined to expand an employer's common law duty to provide a safe working environment to encompass a smoke-free working environment.[65]

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62. *Id.* at 4.

63. 138 Cal. App. 3d 290, 188 Cal. Rptr. 159 (1982).

64. In fact, no link has been shown between ETS and chronic adverse health effects. Both the Surgeon General and the National Academy of Sciences found that available studies preclude any firm conclusion about the relationship between exposure to ETS and cardiovascular disease and that further studies are needed to determine whether any real link exists. *See, e.g.*, SURGEON GENERAL'S REPORT, *supra* note 12, at 14; NAS REPORT, *supra* note 12, at 11. In addition, the Surgeon General's report concluded that "a previously healthy individual would not develop chronic lung disease solely on the basis of involuntary tobacco smoke exposure in adult life." SURGEON GENERAL'S REPORT, *supra* note 12, at 62. Finally, both the Surgeon General and the National Academy of Sciences reports emphasize critical limitations on their finding of a possible connection between exposure to ETS and lung cancer. Upon reviewing the same evidence considered by the NAS and the Surgeon General, the International Agency for Research on Cancer of the World Health Organization concluded, also in late 1986, that the available evidence is equally consistent with the finding of an increase in risk or an absence of risk. IARC MONOGRAPH, *supra* note 12, at 308. A number of other studies published since 1986 also contradict the limited findings of the Surgeon General and NAS reports with respect to the purported relationship between ETS and lung cancer. *See, e.g.*, PROCEEDINGS, *supra* note 12, at 242-50, 252-58.

65. In 1986, a Massachusetts court rejected a nonsmoker's claim against her employer based on breach of contract, and intentional and negligent infliction of emotional distress. Bernard v. Cameron & Colby Co., 397 Mass. 320, 491 N.E.2d 604 (1986). But, in McCarthy v. Washington, 110 Wash. 2d 812, 759 P.2d 351 (1988), the Washington Supreme Court recently held that an employee who allegedly developed lung disease as a result of exposure to tobacco smoke in the workplace was not preempted by workers' compensation laws from stating a cause of action against her former employer for negligence—a finding inconsistent with existing labor law precedents. While four justices opined in dicta that employers have a common law duty to provide a smoke-free work environment, that conclusion was specifically rejected by a majority of the court. *See id.* at 826, 759 P.2d at 358 (Brachtenbach, J., dissenting). In a statement issued by the Washington State Attorney General's office, a spokesperson for the attorney general said that the dicta of the three justices in *McCarthy* did not establish binding law on the issue of an employer's duty to provide a "reasonably safe" workplace. 26 GOVERNMENTAL EMPLOYMENT

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Indeed, several courts that have addressed the issue to date have recognized the need to consider the interests of both smokers and nonsmokers.[66]

## III. Workplace Smoking Legislation

In addition to analyzing case law, the workplace smoking issue demands a careful review of relevant state and local legislation.[67] Thirteen states to date have enacted legislation specifically regulating smoking in private workplaces.[68] These are: (1) Connecticut;[69] (2) Florida;[70] (3) Iowa;[71] (4) Maine;[72] (5) Minnesota;[73] (6) Montana;[74] (7) Nebraska;[75] (8) New Hampshire;[76] (9) New Jersey;[77] (10) Rhode Island;[78] (11) Utah;[79] (12) Vermont;[80] and (13) Washington.[81]

These state laws vary greatly, especially in the degree to which they attempt to displace the role of the employer and its employees in resolving workplace smoking issues. However, virtually all of the state laws have the following common features:

(1) None of the state laws entirely bans smoking in the workplace;[82]

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Relations Report 1172 (Aug. 1988). A trial on the merits in *McCarthy* is scheduled for October 1989.

66. *See* Shimp v. New Jersey Bell Tel., 145 N.J. Super. 516, 531, 386 A.2d 408, 416 (1976); Gordon v. Raven Sys. & Research, 462 A.2d 10, 15 (D.C. App. 1983); *McCarthy*, 110 Wash. 2d at 821-22, 759 P.2d at 355-56.

67. There is no federal legislation regulating smoking in private workplaces. The United States government has, however, adopted smoking restrictions covering the 6,800 buildings controlled by the General Services Administration. These restrictions apply to approximately 890,000 federal employees. *See* 41 C.F.R. Part 101-20.105-3. In addition, the Federal Labor Relations Authority has recently restricted the ability of several federal agencies to change smoking policy without first negotiating with bargaining units. *Treasury Employees Union Chapter 250*, 33 FLRA No. 8, 61-74 (Before Calhoun and McKee) (Oct. 13, 1988), Nos. 0-NG-1524, 0-NG-1536 and 0-NG-1545.

68. This figure represents only those states with laws specifically aimed at private sector workplaces. It does not include the various laws regulating smoking by public sector employees in government-owned buildings or those public safety or occupational regulations pertaining to smoking.

69. Conn. Gen. Stat. Ann. § 31—40q (West 1987 & Supp. 1988).

70. Fla. Stat. Ann. § 386.202—386.209 (West 1986).

71. Iowa Code Ann. § 98A.1—98A.6 (West Supp. 1988).

72. Me. Rev. Stat. Ann. tit. 22, § 1580-A (Supp. 1988).

73. Minn. Stat. Ann. §§ 144.411—144.417 (West Supp. 1989).

74. Mont. Code Ann. §§ 50-40-101—50-40-109 (1987).

75. Neb. Rev. Stat. §§ 71-5701—71-5713 (1986).

76. N.H. Rev. Stat. Ann. §§ 155:50—155:53 (Supp. 1988).

77. N.J. Stat. Ann. ch. 184, §§ 26:3D-23—26:3D-31 (West 1987).

78. R.I. Gen. Laws §§ 23-20.7-1—23.20.7-7 (Supp. 1988).

79. Utah Code Ann. §§ 76-10-101, 76-10-106, 76-10-108—76-10-110 (Supp. 1988).

80. Vt Stat. Ann., tit. 18, §§ 1421—1428 (Supp. 1988).

81. Wash. Rev Code Ann § 70.160.010—70.160.900 (West Supp 1988).

82. In a recent election in Oregon, Proposition 6—which would have imposed a vir-

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(2) Most state laws do not prohibit smoking in specific areas;
(3) Most provide exceptions for enclosed private offices; and
(4) Most provide for only minor penalties, such as minimal fines, for violations.

Local city, county, or other municipal ordinances may also restrict smoking in private workplaces.[83] Local ordinances are often more restrictive and specific than state statutes.[84] Local ordinances may specifically limit smoking in particular areas of the workplace. Some of these local laws, such as the San Francisco, California ordinance, also accord preferential rights to nonsmoking employees within their work area.

Workplace smoking laws can be generally categorized into three groups, according to the degree to which they intrude upon an employer's discretion. These categories are: (1) the "least intrusive" laws, (2) the "partially intrusive" laws, and (3) the "most intrusive" laws.

The least intrusive laws are relatively straightforward. They merely require the employer to establish, post and implement a written policy regarding smoking in the workplace. These laws leave the specifics to the individual employers and do not dictate the content of the policy. For example, New Hampshire's workplace smoking law merely states:

> An employer shall, within 6 months of the effective date of this subdivision, establish and implement written rules governing smoking and nonsmoking in the workplace. The rules shall be readily available for viewing by the employees and may include the designation of smoking and nonsmoking areas. Upon request, the employer shall provide a copy of the written rules to any employee.[85]

Least intrusive laws can also be found in Connecticut, Maine and Montana.

The partially intrusive laws are somewhat more specific. They typically spell out the particulars that employers must include in smoking policies, and indicate the types of efforts required of an employer to accommodate nonsmoking employees. Often they indicate a presumption that smoking should be prohibited in all areas *except* where it is specifically permitted.

Many partially intrusive laws require the employer to allocate smoking and nonsmoking areas based on the numbers of smokers

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tual ban on smoking in all public places, including places of work—was defeated by a three to two margin.

83. Hundreds of local ordinances impact smoking in the workplace. The majority of these are located in California. BNA, SPECIAL REPORT, *supra* note 1, at 66.

84. In Florida and Oklahoma, state law preempts all local smoking ordinances.

85. N.H. REV. STAT. ANN. § 155:52(I) (Supp. 1988).

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and nonsmokers. In addition, some state laws require employers to place physical barriers or "buffer zones" between smoking and nonsmoking employees. States with partially intrusive laws include Florida, Minnesota, Nebraska and Utah.

The most intrusive laws impose significantly greater burdens on employers and limit management's discretion. These laws generally dictate which portions of the workplace must be designated as no-smoking areas. Also, these laws usually specify the percentage of space in employee cafeterias, lunchrooms and lounges that must be reserved for nonsmokers. In some instances, these most intrusive laws give nonsmoking employees superior rights over smokers. For example, the local ordinance in Palo Alto, California, provides that "[i]n any dispute arising under the policy, the rights of the nonsmoker shall be given precedence."[86]

### *A. Objections to Legislation Concerning Workplace Smoking*

The primary and most obvious objection to legislative enforcement of workplace smoking is that it usurps the ability of employers to deal individually with employee concerns as they arise. Legislation does not add to management's bevy of rights; rather, such legislation takes away the considerable flexibility employers have historically enjoyed in this area. In addition, there are numerous other potential objections to legislation covering workplace smoking.

First, smoking laws, like workplace smoking policies, can be difficult to enforce. Regardless of whether this responsibility is placed on the employer or some governmental body, such as a law enforcement agency or local health department, limited resources and personnel makes rigorous enforcement of workplace smoking laws unlikely. Second, because workplace smoking is generally considered to be a *mandatory* subject of bargaining,[87] legislative restrictions may create conflicts between employers and unions. Third, restrictions imposed by smoking legislation may interfere with worker efficiency. For example, where compliance with the law requires employers to rearrange work areas or segregate smoking and nonsmoking employees, an employer's operations may be disrupted and productivity decreased.[88] Fourth, smoking

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86. Palo Alto Municipal Code, ch. 9.14, effective Feb. 1, 1984.

87. *See infra* note 95.

88. According to one study, of those employers polled which have implemented smoking restrictions, seven percent responded that their policies have had a notable effect on company costs. Eight percent indicated that employee productivity increased, while three percent reported that their restrictions had a detrimental effect on employee productivity. BNA, SPECIAL REPORT, *supra* note 1, at 22-23. For an economist's view of the "social costs" of smoking, including lost production, workplace efficiency and absenteeism,

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laws which give nonsmokers unlimited power to dictate the company policy for all workers are likely to be perceived as unfair by others, especially employees who smoke. For instance, a local ordinance in San Francisco allows even one employee to veto an employer's smoking policy in that employee's "office workplace."[89] These types of policies will undoubtedly create resentment among some employees and foster the perception that employers unfairly favor nonsmokers.[90] Fifth, where legislation requires employers to physically separate smokers and nonsmokers, employers may experience a loss of managerial freedom. Finally, some state or local laws regulating smoking in the workplace are likely to be challenged on constitutional or other grounds, and may thus embroil employers in resulting litigation.[91]

As a result of these problems, smoking in the workplace is often best left to the discretion of management and its employees. Excessive regulation in this area substantially undermines an employer's ability to respond flexibly to smoking-related problems, while at the same time it creates a morass of practical and legal problems. Moreover, smoking ordinances are not a panacea. They cannot take into account the peculiarities of each workplace, nor can they address the unique problems and personalities involved in any given situation. Employers must still resolve the competing interests of smoking and nonsmoking employees.

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*see* R. TOLLISON & R. WAGNER, SMOKING AND THE STATE ch. 3 (1988).

89. San Francisco Municipal Health Code, Smoking Pollution Control Ordinance No. 298-83 (Proposition P), effective Mar. 1, 1984.

90. Thus far, employee sentiments about smoking policies have been mixed. Overall, 42% of recent survey respondents said the smokers think their policy is "about right," while 33% indicated the rules were too restrictive; 53% of firms with policies said that nonsmokers were satisfied, while 33% said nonsmokers wanted tougher restrictions. BNA, SPECIAL REPORT, *supra* note 1, at 23.

Press accounts of employee sentiment are decidedly mixed as well. *See Do You Smoke? Drink? If So, Some Employers Say, You May as Well Stay Home*, BUSINESS FIRST-COLUMBUS, vol. 3, no. 36, § 2, at 3; *The Company Is Watching You Everywhere*, N.Y. Times, Feb. 15, 1987, § 4, at 21, col. 2, (Editorial Desk); *Some Workers Upset by Company Smoking Ban*, AP, Jan. 21, 1987; *Bans, Red Ink: Smoking: A Burning Issue*, L.A. Times, Nov. 21, 1985, § 1, at 1, col. 1, (Metro Desk); *Where There's Smoke, There's Ire: After Years on the Defensive, Smokers Fight Back*, L.A. Times, Jan. 14, 1988, § 4, at 1, col. 1.

91. The primary challenge is that smoking laws are often too vague to set definite standards of compliance for employers. Indeed, several smoking ordinances have already been struck down as unconstitutionally vague. *See, e.g.*, Florida v. Burton, No. 80-999CO-A-42 (Fla. Cir. Ct. 1981) (ordinance restricting smoking in public places unconstitutionally vague); Greater Rockford Food Servs. v. Orthoefer, No. 76-2447 (Ill. Cir. Ct. 1976) (ordinance restricting smoking in public place struck down as unduly vague and in violation of constitutional guarantee of equal protection).

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## IV. Labor Relations Issue

Unionized employers seeking to adopt workplace smoking policies, including policies necessitated by statute or ordinance, must consider whether such policies can be adopted unilaterally without bargaining with the union and whether the policy violates the collective bargaining agreement.[92]

The National Labor Relations Act[93] (NLRA) prohibits an employer from unilaterally changing the terms and conditions of employment without bargaining with the union representing the employees. Failure to do so constitutes an unfair labor practice under section 8(a)(5) of the NLRA.[94]

Rules governing workplace smoking have been held to be "terms and conditions of employment" and, accordingly, are subject to mandatory collective bargaining.[95] Thus, unless the collective bargaining agreement includes a broad "management rights" clause[96] permitting the employer to unilaterally establish plant rules, the employer must first bargain with the union prior to restricting smoking in the workplace.[97] This is especially true in sit-

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92. In 1986, the AFL-CIO issued a National Resolution opposing unilateral attempts by management to impose workplace smoking policies. It provides, in part:

> Unions are faced with legislation or unilaterally imposed employer policies that forbid smoking on the job and infringe on the rights of workers who smoke. Unions have a legal responsibility to represent the interests of all their members—smokers and *nonsmokers*. The AFL-CIO believes that issues related to smoking on the job can best be worked out voluntarily in individual workplaces between labor and management in a manner that protects the interests and rights of all workers and not by legislative mandate.

*See* BNA, Special Report, *supra* note 1, app. D at 2. There have also been scattered reports that rules regarding smoking in the workplace have become issues in union organizing drives.

93. 29 U.S.C. §§ 151-169 (1982).

94. 29 U.S.C. § 158(a)(5) (1982). The National Labor Relations Board has broad powers to prevent and/or remedy unfair labor practices. *See generally* 29 U.S.C. § 160 (1982).

95. *See* Chemtronics, Inc., 236 NLRB No. 21 (1978). *See also* Pennsylvania v. Pennsylvania Labor Relations Bd., 74 Pa. Commw. 1, 459 A.2d 452 (1983) ("[t]he subject of whether employees may smoke at their workplaces appears to be at the center of those subjects properly described as 'conditions of employment' "). *See also* Gallenkamp Stores v. NLRB, 402 F.2d 525 (9th Cir. 1968); S. S. Kresge v. NLRB, 416 F.2d 1225 (6th Cir. 1969); Wintergarden Citrus Prod. v. NLRB, 238 F.2d 128, 129 (5th Cir. 1956); NLRB v. Hilton Mobile Homes, 387 F.2d 7, 10-11 (8th Cir. 1967).

96. An employer may insist upon a broad "management rights" clause. *See* NLRB v. American Nat'l Ins. Co., 343 U.S. 395 (1952). In general, such clauses give employers considerably more discretion over specific aspects of employment, which may include promotions, transfers, plant rules, etc. It is significant that the NLRB General Counsel has recently issued guidelines stating that employers must bargain with their unions before instituting any drug-testing policy. *See* NLRB Memorandum GC 87-5 (Sept. 8, 1987). The NLRB may adopt a similar position regarding smoking policies.

97. To date, no court has found a management rights clause sufficiently broad to permit the unilateral imposition of a smoking policy. In Pennsylvania v. Pennsylvania Labor Relations Bd., 74 Pa. Commw. 1, 459 A.2d 452 (1983) the court rejected an em-

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uations where smoking is a recognized right or privilege of employment.

This issue was discussed in *In re Parker Pen U.S.A.*[98] In this case, the employer—who had permitted on-the-job smoking for over twenty years—unilaterally abolished employee smoking rights that were guaranteed under the collective bargaining agreement. The employer allegedly did so for health reasons after receiving the Surgeon General's 1986 report on involuntary smoking.[99] In resolving an employee grievance, the arbitrator held that "both parties have an interest in addressing the profound issue raised by the employer concerning the safety of the workplace."[100] Accordingly, the arbitrator invalidated the employer's smoking ban until any changes could be bargained over during upcoming negotiations.

Even where the employer is required to impose smoking restrictions pursuant to state statute or local ordinance, it should bargain over all discretionary aspects of the rule.[101] In practice, bargaining will be routinely required because most workplace smoking laws leave a considerable amount of discretion to employers.[102]

Even if the unilateral implementation of a smoking policy does not violate the NLRA, it may nonetheless violate the collective

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ployer's argument that it had the "inherent managerial" authority to determine unilaterally whether to permit smoking at employee work stations. In the arbitration setting *compare* Ohio Dept. of Health, 89 Lab. Arb. (BNA) 937 (1987) (Cohen, Arb.); Morelite Equip. Co., 88 Lab. Arb (BNA) 777 (1987) (Stoltenberg, Arb.); Saap-On Tools Corp., 87 Lab. Arb. (BNA) 785 (1986) (Berman, Arb.); National Pen & Pencil Co., 87 Lab. Arb (BNA) 1081 (1986) (Nicholas, Arb.); Litton Indus., 75 Lab. Arb. (BNA) 308 (1980) (Grabb, Arb.); and Sherwood Medical Indus., 72 Lab. Arb. (BNA) 258 (1977) (Yarowsky, Arb.) (smoking restrictions upheld) with Dental Command, Dept. of the Army, 83 Lab. Arb. (BNA) (Allen, Arb.) 529 (1984); Union Sanitary Dist., 79 Lab. Arb. (BNA) 193 (1982) (Koven, Arb.); and Schien Body & Equip. Co., 69 Lab. Arb (BNA) 930 (1977) (Roberts, Arb.) (smoking restrictions invalid).

98. 90 Lab. Arb. (BNA) 489 (1987) (Fleischli, Arb.).

99. *See* SURGEON GENERAL'S REPORT, *supra* note 12.

100. 90 Lab. Arb. at 496.

101. There are no "smoking policy" cases directly on this point. The institution of a smoking policy would constitute a "term and condition" of employment and, therefore, be a mandatory subject of bargaining. *See supra* note 95 and cases cited therein. Nevertheless, neither union nor management may require the other to agree to provisions that are unlawful or prohibited. Meat Cutters Local 421, 81 N.L.R.B. 1052 (1949); Borg-Warner v. NLRB, 356 U.S. 342 (1958). Thus, proposed or existing provisions that directly conflict with legislation automatically become illegal or unenforceable. Hughes Tool Co., 147 N.L.R.B. 1573 (1964); Savannah Printing Specialties & Paper Prod. Local 604 v. Union Camp Co., 50 F. Supp. 632 (S.D. Ga. 1972). Legislation that provides employers with discretion, however, such as that which simply requires employers to "adopt" a smoking policy, would not be affected. That is, the particular discretionary aspects of the policy would still be a mandatory subject of bargaining.

102. For example, the New Hampshire law cited at *supra* text accompanying note 85, merely directs the employer to adopt a smoking policy. *All* of the specifics are left to the discretion of each individual employer.

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bargaining agreement. Arbitral decisions have consistently stated that to be valid, an employer rule must be reasonable under the circumstances and nondiscriminatory in application.[103] Arbitrators have struck down employer smoking policies that fail to meet this standard.

In *Union Sanitary District*,[104] the arbitrator found that the employer could not unilaterally prohibit employees from smoking in their offices. Specifically, the arbitrator found that the absolute prohibition was arbitrary because there was no adequate basis for the rule. Although the employer stated it wanted to protect nonsmoking employees, the evidence showed there were only two bargaining unit members in the building who smoked; for six hours a day they were not in their offices but were out in the field; and no one complained about the smoking. Moreover, the California Indoor Clean Air Act of 1976,[105] which the employer cited to justify its ban, did not require a ban on smoking. Rather, that Act contemplates a relatively flexible regulation of smoking which recognizes the rights of both smokers and nonsmokers.[106]

## V. How Should Employers Respond to the Workplace Smoking Issue?

Each employer's reaction to the workplace smoking issue will, naturally, depend on the needs and circumstances of its workplace. It is important in any situation to encourage a spirit of cooperation and communication among employees and management. Indeed, a recent survey of 1,100 employers indicated that over seventy percent expect employees to address workplace smoking issues among themselves before invoking management's time and

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103. *See* United Tele. Co. of Florida, 78 Lab. Arb. (BNA) 865 (1982)(designation of no-smoking table in cafeteria upheld as reasonable in light of company and union's interests in maintaining a healthy work environment and minimizing expenses and potential liability); H-N Advertising & Display Co., 88 Lab. Arb. (BNA) 329 (1986), 88 Lab. Arb. (BNA) 1311 (1987)(rule banning smoking in area of plant where combustibles are stored was reasonable and nondiscriminatory where worker safety was primary reason for expanding rule and implementation of measures to improve safety is normally management prerogative). *See also supra* note 97.

104. 79 Lab. Arb. (BNA) 193 (1982) (Koven, Arb).

105. Cal. Health & Safety Code § 25940-25947 (Deering 1988).

106. *See also* Schlen Body & Equip. Corp., 69 Lab. Arb. (BNA) 930 (1977) (Roberts, Arb.) (employer plant-wide smoking ban unreasonable because there was no proof that the rule clearly benefitted nonsmokers since work area was well ventilated, nor was there any indication that the ban directly improved workers' health). *But cf.* Ohio Dept. of Health, 89 Lab. Arb. (BNA) 937 (1987) (Cohen, Arb.) (state's modified smoking policy for health department employees found reasonably related to legitimate objectives); Morelite Equip. Co., 88 Lab. Arb. (BNA) 777 (1987) (Stoltenberg, Arb.) (smoking ban at workstations reasonable in view of fire dangers); Litton Indus., 75 Lab. Arb. (BNA) 308 (1980) (Grabb, Arb.) (rule limiting smoking to specific areas reasonable).

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efforts.[107] Employers should also consider the following specific issues:

(1) Is there an open line of communication so that employees can effectively express their concerns and thereby informally resolve smoking-related disputes?

(2) Have employees complained about co-workers' smoking or tobacco smoke in the work environment?

(3) If employees have complained, how many have done so, and on what basis? Are such complaints properly attributable to employee rivalries or individual medical hypersensitivity(ies)?

(4) Is poor ventilation the cause of actual or perceived indoor air quality problems?[108]

(5) Would the imposition of smoking restrictions in the workplace decrease productivity, adversely affect employee morale, violate the provisions of a collective-bargaining agreement or give rise to discrimination claims?

(6) Could smoking disputes be resolved by management through less drastic means, such as separating smokers from nonsmokers, erecting partitions or improving the company's ventilation system?

After evaluating the work environment based on the foregoing considerations, and addressing the likely accommodation options, an employer should decide whether it is necessary to implement a formal smoking policy.[109]

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107. HUMAN RESOURCES POLICY CORP., SMOKING POLICIES IN LARGE CORPORATIONS (May 1988) [hereinafter SMOKING POLICIES]. *See also* BNA, SPECIAL REPORT, *supra* note 1, at 26 figure E (72% of surveyed employers urged employees to resolve smoking-related problems themselves).

Other employer responses to complaints of workplace smoking included: (1) attempt to get smoker to reduce smoking (22.5%); (2) do nothing (9.7%); (3) move complainer to new work area (6.3%); (4) move smoker to new work area (3.4%); (5) other measures (3.1%); and (6) order smoker to discontinue smoking (0.9%). SMOKING POLICIES, *supra*, at 12, Table 13. *See also* BNA, SPECIAL REPORT, *supra* note 1, at 26.

108. There is substantial evidence that air quality complaints are indicative of a much larger problem, i.e., inadequate ventilation. For example, a January 1987 report prepared by the National Institute for Occupational Safety and Health (NIOSH) attributed 52% of complaints connected to indoor air quality to "inadequate ventilation." Only 17% of the complaints were attributable to indoor contaminants, including tobacco smoke (which accounted for only 2%). In addition, chemicals emitted from carpeting, furniture and copying machines also contribute to indoor air contamination. BNA, SPECIAL REPORT, *supra* note 1, at 9-10. Similarly, according to a report entitled *Source Nature and Symptomology of Indoor Air Pollutants* prepared by ACVA, Atlantia, Inc., a Fairfax, Virginia company specializing in the study and assessment of indoor air pollution, environmental tobacco smoke was found to be the immediate cause of indoor air problems in only four percent of the 233 major buildings investigated between 1981 and 1987. ACVA, ATLANTIA, INC., SOURCE, NATURE AND SYMPTOMOLOGY OF INDOOR AIR POLLUTANTS 9-11 (1987).

109. A majority (63.8%) of the 1,100 corporations who responded to the SMOKING POLICIES survey had not adopted any formal smoking policy. The survey also found that the companies most likely to have smoking policies are geographically located in areas with

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## VI. Drafting A Smoking Policy

In jurisdictions where workplace smoking is governed by a state statute or local ordinance, employers must conform their policies and practices to the law. In some circumstances, this may require employers to adopt a formal smoking policy. In the vast majority of jurisdictions, however, employers are still free to decide whether a smoking policy is necessary or appropriate. In doing so, employers may want to evaluate whether there is a predicate for action. In this regard, they may find it useful to survey their employees to see if there is a consensus of opinion. Management may also want to consult its labor unions, if any.

Should a company decide that a formal written policy is necessary, the specifics of the policy will naturally depend upon the individual aspects of the workplace. Because of local differences, particularly in those companies with decentralized decision making, some companies have developed a smoking policy applicable to only some divisions, offices or plants. Other employers have adopted a smoking policy in response to a specific problem or where they are governed by a particular local ordinance. In addition, companies tend to vary their smoking policies depending upon the degree of specificity desired. A less specific smoking policy aimed at promoting cooperation and consideration might, for example, state:

> It is our policy to make every reasonable effort to accommodate all employees within the constraints imposed by our physical structure and financial resources. It is our firm conviction that the wishes of smokers and nonsmokers can best be resolved through cooperation, dialogue and common courtesy. Should a dispute or concern arise, management and employees should work together to seek a reasonable resolution consistent with this policy.

A nonspecific policy such as this will increase flexibility and allow management to resolve individual disputes on a case-by-case basis.

In contrast, some employers may opt for a smoking policy with a greater degree of specificity. For instance, the employer may want to designate particular smoking or nonsmoking areas or workstations. The specific locations covered may include: private offices, hallways, conference rooms, lunch rooms, restrooms and auditoriums.[110]

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workplace smoking laws. Smoking Policies, *supra* note 107, at 4. More recently, a smaller BNA survey of 623 employers indicated that a minority (43%) of employers have not adopted any workplace smoking policy. BNA, Special Report, *supra* note 1, at 2.

110. If enforced in an arbitrary or discriminatory manner, a smoking policy may subject an employer to potential liability. Indeed, inequitable enforcement could foster employee discontent and possibly support claims premised on breach of contract or tort claims

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Smoking bans, while rare, pose more serious problems.[111] This is especially true if they proscribe off-duty behavior. In addition to employee morale problems, these bans are likely to give rise to a morass of legal claims.[112] For these reasons, employers should be extremely cautious before considering a total ban on workplace smoking.

## CONCLUSION

Smoking can be the subject of emotional debate in the workplace. There are few uniform answers to guide management in dealing with the issue. But, left with enough flexibility to address the concerns of all of their employees, most employers generally find that they can resolve smoking disputes by undertaking practical accommodations on a case-by-case basis.

Some specific options for resolving smoking disputes include: (1) separating smokers from nonsmokers; (2) moving nonsmokers closer to windows or fresh air ducts; and (3) improving ventilation throughout the workplace. However difficult and legally complex the smoking in the workplace issue has become, one thing is clear: Employers have an obligation to accommodate the competing interests of smoking and nonsmoking employees. It is equally clear that in an era of increasing labor shortages, employers are redoubling their efforts to select and retain skilled and experienced workers—smokers as well as nonsmokers.

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against employers or individual supervisors. *See* Carroll v. Tennessee Valley Auth., 697 F. Supp. 508 (D.D.C. 1988) (public employer not shielded from potential tort liability under "official immunity" doctrine, because supervisor who failed to enforce smoking policy acted outside course and scope of his employment). In *Carroll*, the plaintiff claimed that she had developed lung disease allegedly from exposure to environmental tobacco smoke on the job. In addition, she alleged that her supervisors took reprisals against her by giving her poor performance evaluations, assigning her demeaning work and questioning the seriousness of her health claims. On November 1, 1988, this case was settled for an undisclosed sum of money.

111. The vast majority of employers with smoking policies do not ban smoking entirely. As noted above, those which do typically do so due to product (food) contamination concerns or because flammable materials are produced or stored in the workplace. And few (five percent) give hiring preference to nonsmoking job applicants. BNA, SPECIAL REPORT, *supra* note 1, at 17, 22. One notable exception pertains to police and fire departments, which are faced with unique workers' compensation issues. *See supra* notes 24-28 and accompanying text. *See also Bans. Red Ink: Smoking: A Burning Work Issue*, *supra* note 90 (Pacific Northwest Bell bans smoking in all facilities; Radar Electric of Seattle will not hire smokers; Capital City Products conducts seminars to help employees quit smoking).

112. For a thorough discussion of smoking bans, see Rothstein, *supra* note 24, at 940.

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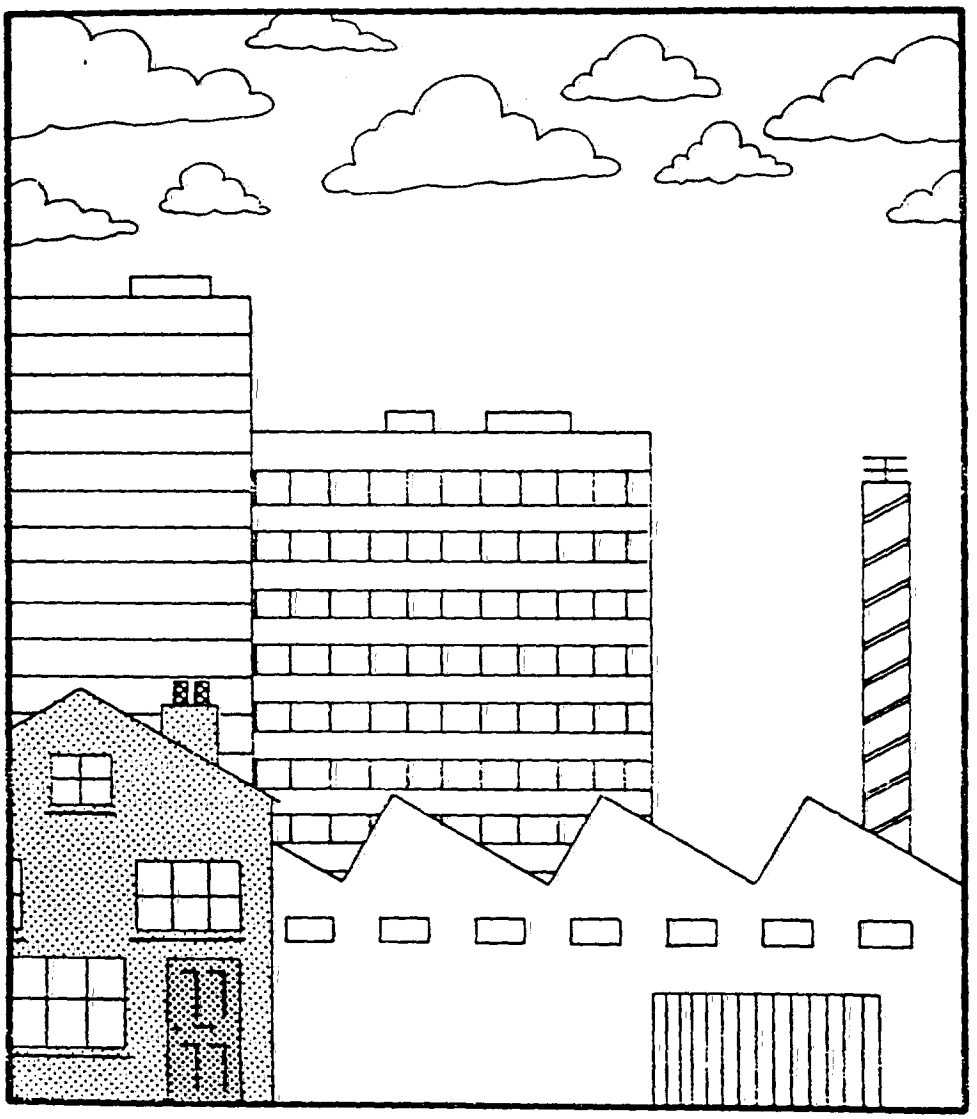
# INDOOR AND AMBIENT AIR QUALITY



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# HEALTH EFFECTS OF PASSIVE SMOKING IN THE WORKPLACE

W.A.Crawford

Consultant in Occupational Health & Preventative Medicine, 29 Battle Boulevarde, Seaforth, NSW 2092, Australia

ABSTRACT

Concern is being induced in the non-smoker subjected to other people's tobacco smoke and concern is being induced in smokers that they disadvantage the health of others. If a ventilation system cannot cope with ETS it will not cope with other unseen environmental hazards. Present scientific data permit only the formation of hypotheses and are insufficient to justify the forming of public policies. The prohibition of smoking in the workplace will not reduce the adverse health effects of industrial processes. It will remove the telltale signs of inadequate ventilation.

## INTRODUCTION

In most workplaces the least of the plausible potential hazards is exposure to environmental tobacco smoke (ETS). The major known hazards dominate the overall scene and for many the American Conference of Government Industrial Hygenists have recommended tolerable limits which, we hope, if achieved, will protect the health of the worker from the adverse effects which are known or hypothesised to occur given time at higher concentrations. In my experience these limits are not often met and, in Australia certainly, are rarely measured. The potential for inducing illness involving the lungs, the cardiovascular system and for the induction, initiation and promotion of cancers is commonly present. In addition to specific carcinogenic chemicals and particulates there are industries in which, while no specific carcinogen has been identified, there has been found epidemiologic evidence of increased relative risks of cancer.

Most industries are found in towns, as are offices and in the towns compared to rural areas there are higher levels of pollutants such as polycyclic aromatic amines usually measured as Benzo-a-pyrene. These originate in the factories, transport systems and dwellings from the use of fossil fuels. Carbon monoxide and particulates are measurable and can reach levels considered to pose a threat to health.

Many workers work in an unhygienic factory or office in a dirty town and live in unhygienic homes and many of them are smokers. Despite these conditions the expectation of longevity continues to improve. This improvement takes many people into the older age groups and thus the cumulative dosage of agents hostile to the respiratory system has increased.

It is with this complex environmental background of pollutants that the health effects of exposure to ETS in the workplace is addressed. It is of note that only two papers located have addressed the subject. They are White and Froeb (1) in Southern California and Kentner (2) in Bavaria. White and

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Froeb (1) studied lung function and acquired suspect results. The Kentner (2) study found no adverse effects. No studies have been located which addressed lung or other cancers in nonsmokers in workplaces where smoking was permitted or was not permitted.

It is of note that the major study of smoking in the workplace and its health effects is that of the US Surgeon General (3) in 1985 and in that 516 page document, ETS receives only four minor mentions and these pertain to the asbestos using industries. The pioneering works published in 1981 on lung cancer and passive smoking by Hirayama (4) in Japan and Trichopoulos (5) in Greece and Garfinkel (6) et al in the USA had some four years earlier proposed serious hypotheses. The hypotheses are still under test in 1988.

## HISTORICAL BACKGROUND

The Surgeon General's (3) 1985 report on smoking and health in the workplace emphasises the opportunities to conduct anti-smoking and smoking cessation campaigns in these relatively closed communities with their relatively captive employees.

There was an immediate hostile response from the AFL-CIO (7), the US Trade Unions organisation which included:

"The Surgeon General's report on The Health Consequences of Smoking - Cancer and Chronic Lung Disease in the Workplace will seriously set back efforts to protect the health of American workers.

"The report minimizes and misrepresents the risks posed by occupational hazards, and suggests that if workers stop smoking they will eliminate the risk of occupational disease. In fact, the way to eliminate occupational disease is by reducing workplace hazards.

"Unfortunately, this latest report adds no new scientific information to our knowledge of the risks posed to workers on the job.

"This report can only hurt workers by undermining efforts to control workplace hazards. It will lead to misdiagnosis of occupational disease. It will make it even more difficult for workers suffering occupationally-related disease to secure compensation to which they are entitled. And it will be used by those who are responsible for poisoning workers to avoid legal liability.

"Issuing a report that is scientifically unsound detracts from efforts of the Surgeon General and others to deal with the serious public health problems of both smoking and occupational disease."

In 1986 two major reports on the health effects of ETS were published. One by the US National Academy of Science (8) and the other by the US Surgeon General (9). Neither of these massive reports references new studies in the workplace. They review the studies of health effects in adults with regard to respiratory function and disease, cardiovascular function and disease and lung cancers in the nonsmoking spouses of active smokers, the spouses were dominantly women. (Their reviews of the effect on children is not relevant to this contribution to the debate on ETS in the workplace).

Basically, from the studies on ETS reported, there is inadequate data on respiratory effects in workers because of the massive and diverse confounding factors in the workplace and general industrial areas. There is even less data on cardiovascular effects, if any at all, in healthy adults. This is the conclusion of the National Academy of Science (8) advisers.

The most potent tool in the hands of those using exposure to ETS to bolster anti-smoking campaigns aimed at the public, the workforce and politicians is the threat of lung cancer induction. The bases for this tool are the epidemiological reports of the US Department of Health (9) 1986, the National Academy of Science/National Research Council (8) 1986, and the International Agency for Research on Cancer (10) 1986. There have also been conflicting personal views expressed, Aviado (11) 1986, Wald (12) 1987, Lebowitz (13) 1986

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and Uberla (14) 1987. Various symposia have been published on the issues, Geneva (15) 1983, Vienna (16) 1984 and Essen (17) 1986. The conclusions reached by these agencies, people and workshops are in conflict.

In these major studies the statistical significances and the confidence limits, with the exception of the Hirayama (4) and Trichopoulos (5) studies, contain the null hypothesis level and despite this some units or persons apply judgments or even wishful thinking to justify anti-smoking policies. Professor Weiss (18) from Harvard University in his review of the USSG report on smoking in the workplace notes:

"The Surgeon General, clearly recognizing that the government cannot regulate what goes on in private homes, is taking the only action available to him in suggesting that work-place exposure be banned. The health impact of exposure at work, except for its irritation, is largely unknown. The report is on its weakest ground scientifically here. However, if the effect of restricting smoking at work is to reduce the total number of smokers then the action would seem justified despite the paucity of scientific data supporting it. Restricting exposure is only part of the solution to this problem. Further research and education are still needed."

Earlier, in 1981, E Culyer Hammond and Irving J Selikoff (19), commenting on the Hirayama and Trichopoulos studies, made a most important observation on the potential flow-on in occupation health and hygiene practices and, again, I quote:

"At first glance, the only practical issue raised by the evidence discussed above is whether or not people should be strongly urged not to smoke on the grounds that in doing so they increase the risk of lung cancer in their fellow citizens (particularly members of their own families). The fact that smoking increases the risk for the smoker is already well known and generally accepted.

"This is an important issue. But there is an even more important issue. If low levels of "passive smoking" increase the risk of lung cancer to a significant degree (as two studies seem to indicate), this strongly suggests that the dose-response curve is approximately logistic in shape, rather than in a straight line. If this is so for one chemical carcinogen (tobacco smoke) and one type of cancer, it is likely to be so for other chemical carcinogens and other types of cancer. This could radically alter present impressions of "tolerable levels of exposure" for a host of other agents.

"With so much at stake, we hesitate to express an opinion at this time on whether "passive smoking" increases the risk of lung cancer to a significant degree. It would take additional positive evidence, but perhaps not a great deal more evidence, to firmly convice us that this is so."

Such evidence has not been presented and there have been few changes in Threshold Limit Values since then.

Hammond and his colleagues Auerbach and Garfinkel (16) in a study of the histological changes in the airways of the lung in 1979 and referred to at a conference in Vienna stated, and again I quote:

" On the 57 nonsmoking cases, virtually all slides had "0%" atypical cells. If passive smoking does indeed lead to lung cancer, it would be expected that some of the nonsmokers who had been exposed to smoke of others would have shown histological changes in their bronchial epithelium similar to those found in the light (less than 10 cigarettes a day) smokers, but no such cases were observed. This does not exclude the possibility that the mechanism by which passive smoking could lead to cancer might be totally different from that in smokers."

Similar findings came from Adelaide in Australia by Bertram (20) et al. Weiss (20) found almost no fibrosis in the lungs of nonsmokers.

There were some other important statements made and reported at that Vienna conference. Dr Wynder (16) raised a question which has not yet been answered:

"If passive inhalation in fact increases our risk of lung cancer, then there should have been a steady increase in the incidence of lung cancer among

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nonsmokers. This should have been observed in the female population for the last 40 years. As more and more men smoke, more women passively inhale the smoke of their husbands. Garfinkel and I agree that there has been no significant increase in the incidence of lung cancer in male or female nonsmokers. In this case we have a nonfit."

Hirayama (16) contributed to the problem by saying:

"To me, "direct passive smoking" means the subject is within a distance of 1 or 1.5 m from the source of exposure. That kind of experimental setup must be possible. The two issues of distance and dose should be differentiated when considering the polluting of room air by tobacco smoke. In actual experience, these two factors act together. This is true in the example of an office worker whose dose will depend on the distance he sits from a heavy smoker. In my opinion, room air contamination may not play a major role in a carcinogenic effect of passive smoking - at least in Japan - because my own data suggest that direct passive smoking is far more important."

Doll and Peto (21) in their assessment of avoidable risks of cancer in the US, commissioned by the Office of Technology Assessment of the US Congress also state independently of Garfinkel:

"Pending further data, we remain conconvinced that any material trends in true lung cancer death rates among American nonsmokers have occurred in recent decades (although some such increases should be expected if the effects of "passive" smoking reported by Hirayama (1981) and Trichopoulos et al (1981) are confirmed). A fortiori, we are even less convinced that any such trends not attributable to passive smoking among nonsmokers have occurred."

The International Agency for Research in Cancer (10) 1986 reviewed the overall biological data and the epidemiological data and concluded:

"Examination of smoke from the different sources shows that all three types contain chemicals that are both carcinogenic and mutagenic. The amounts absorbed by passive smokers are, however, small, and effects are unlikely to be detectable unless exposure is substantial and very large numbers of people are observed. The observations on nonsmokers that have been made so far are compatible with either an increased risk from 'passive' smoking or an absence of risk. Knowledge of the nature of sidestream and mainstream smoke, of the materials absorbed during 'passive' smoking, and of the quantitative relationships between dose and effect that are commonly observed from exposure to carcinogens, however, leads to the conclusion that passive smoking gives rise to some risk of cancer."

## WHAT ARE THE KNOWN AND SUSPECTED COMPETITORS IN THE WORKPLACE AND INDUSTRIAL AREAS?

The Doll and Peto (21) report lists the established occupational causes of cancer for lung as arsenic, asbestos, bischloromethyl ether, chromium, ionising radiations, mustard gas, nickel and polycyclic aromatic hydrocarbons. Occupations with raised Standardised Mortality Ratios for lung cancer are also tabled: Miners and quarrymen, gas, coke and chemicals makers, glass and ceramic makers, furnace, forge, foundry, rolling mill workers, electrical and electronic workers, engineering and allied trades workers n.e.c., woodworkers, leather workers, clothing workers, food, drink and tobacco workers, construction workers, painters and decorators, drivers of stationary engines, cranes, etc., labourers n.e.c., transport and communication workers, warehousemen, storekeepers, packers, bottlers, and service, sport and recreation workers.

In the occupational oriented medical journals 1985-87 lung cancer increased risks have been reported for exposure to formaldehyde, wood dust, acrylonitrile, carbon black, benzene, diesel exhaust, wood and chemicals and man made vitreous fibres and for rubber workers, smelter workers, chromium platers, leather workers, bakers and cooks. Variables which require

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consideration listed by the NRC (8) are occupation and industry of employment, exposure to specific respiratory carcinogens, such as asbestos, arsenic, radon, etc., in occupational or non-occupational settings, dietary factors, family history of cancer, residential history, housing characteristics, years of education and socioeconomic status.

Doll (22), referring to the urban-rural lung cancer rate gradient, expresses the view that the differences were not generally large, being commonly about two-fold but that it was notable that mortality increased progressively with the size of the town. He refers also to specific carcinogenic agents in the urban air environment such as POMs, asbestos, arsenic and radon. He also notes the substantial differences in sex incidence between males and females in all countries studied and the wide range in rates from country to country. As example, Scotland had age standardised lung cancer rates per 100,000 of 78.14 in males to 11.71 in females while Portugal had 10.91 and 2.74 respectively. A similar wide range of rates in towns is reported from the polluted to not so polluted areas.

The two-fold increase in rate has been noted by Curwen (23) et al 1954, Haentzel (24) 1961, Prindle (25) 1959, Levin (26) et al 1960, and Buell and Dunn (27) 1967 (Cited by Carnow (28)). The majority of the studies were controlled for smoking effects.

Doll and Peto (21) 1981, conclude their evaluation of environmental exposure to BaP with the assessment that, "it is unlikely to account for more than 1% (say, 1,000) of the US cases of lung cancer in the future". What of the past exposure which affects us now?

That lung cancer is more common in urban areas does not by itself give evidence sufficient to infer that urban air pollution is the attributable factor for lung cancer. There are confounding factors in urban living related to smoking, drinking, eating habits, work, lifestyle, indoor pollutants from heating and cooking and familial inheritance.

Carnow (28) also notes the evidence from studies of migrants which add weight to the hypothesis that air pollution in industrial areas is related to lung cancer, e.g., migrants from Britain going to relatively less polluted countries such as South Africa, Australia and New Zealand. The Britain - South Africa migrant lung cancer changes are described by Dean (29) 1959, the Britain - Australia also by Dean (30) 1964 and Britain - New Zealand by Eastcott (31) 1956.

In contrast to the extreme paucity of reports of investigation into the effect of ETS in workplaces and an absence of extrapolations from the data on active smoking in the asbestos industry and industries using asbestos, silicious materials, coal, cotton dusts etc., there is considerable data presented on air conditioned offices and buildings.

The Sick Building Syndrome has emerged and with it the finding of many indoor air pollutions as competitors to ETS in the health effects market. These competitors include airborne phenolics, asbestos, carbon monoxide, carbon dioxide, plastics, paints, ozone, solvents, disinfectants, formaldehyde, trichlorethylene and other gasses and particulates. Some others penetrate the building from the generally polluted air environment of the city streets and inevitably contain polycyclic aromatic hydrocarbons from motor vehicle exhausts. The latter, like ETS, is known to contain low concentrations of carcinogens.

The syndrome has been described variously but basically consists of complaints of eye irritation, dry mouth and throat, nasal irritation, lethargy, headache, stuffiness and vague general feelings of ill-health.

In England reports by Robertson (32) et al and Finnegan (33) et al indicate that it is a problem of air conditioned buildings more so than naturally ventilated buildings. In the USA and Canada there are government reports from the National Institute of Occupational Safety and Health (34) and Health and Welfare Medical Services (35) on the possible origin or origins of the

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complaints. Interestingly, complaints of ETS contributed only 2% to the suspected problems. Inadequate ventilation was the dominant complaint having a 50% problem rating. Professor Sterling (36) from Canada, a pioneer in the area, will speak on this subject at this meeting. From Honolulu a report by Kodama and McGee (37) specifically reports on biological pollutants and the adverse findings in air conditioned premises.

Here again are data which do not seriously incriminate ETS as a hazard commonly encountered and, when it is, control by ventilation is the answer. If that ventilation cannot control ETS it cannot control the other pollutants as indicated by the AFL/CIO (7) attitude noted above. In addition the presence of radon and radon daughters in buildings which have carcinogenic effects on the lung is now actively being researched in the US, Europe, UK and Australia.

When the NAS (8) aggregated the US studies a relative risk for lung cancer in the spouses of smokers was 1.14 with confidence limits of 0.92-1.40; how is this to be translated? More importantly, how can these measures be extrapolated for the general public and workers in industrial areas?

Doll and Peto (21) indicated in their 1981 study in the US that relative risks of less than 2 are difficult to interpret. Rose (38) in UK also notes that in a multifactorial disease a factor that increases the risk by less than half will almost certainly be undetectible. In a study of cancers in New Zealand, Pearce from the US and Howard from New Zealand (39) restricted their interests to those cancers with relative risks of 1.5 or more as such small risks are particularly likely to be due to bias or confounding.

## CURRENT STATUS AND CONCLUSIONS

The overall scenario in 1988 is that the effects of ETS on the health of workers in industry has received virtually no investigations. The blue collar nonsmoking worker is probably exposed to more ETS than white collar workers as the former tend to have more smoking co-workers than the latter. In these industries recognised as posing threats to the respiratory tract from fumes, dusts, fibres and gasses no specific data has been located.

There has been an upsurge in research into the environmental conditions in offices and buildings and health effects of a minor nature are frequently observed and are clearly multifactorial.

Studies on the excretion of the nicotine metabolite cotinine in the UK and USA indicate that nonsmokers absorb ETS to a minor extent and it is plausible that they absorb other chemicals to the same degree.

Smokers are exposed to mainstream smoke, are exposed directly to sidestream smoke and to the highly diluted and aged ETS and excrete relatively large quantities of cotinine compared to nonsmokers. The differences in quality and quantity of the smoke inhaled is such that extrapolations from the epidemiological data on smokers is fraught with uncertainties.

Some nonsmokers find ETS to be irritating to mucosae, particularly to those of the eyes, but usually exposure to a high concentration in a poorly ventilated area is required.

Thus we are at the stage in 1988 when the only scientific data are:

1. That no atypical cellular changes have been found in the lungs of nonsmokers.
2. That nonsmokers do absorb one chemical peculiar to cigarette smoke - nicotine and this is measured as the metabolite cotinine.
3. There are competitors in the workplace for the induction of lung cancer.
4. There are competitors for lung cancer in air pollution in industrialised countries.
5. There is no evidence of increase of lung cancer in nonsmokers during the decades of increasing prevalence of smoking.
6. There is inadequate data on the epidemiology of ETS in the workplace.

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It is therefore unreasonable in a workplace anti-smoking program to use the threat of respiratory and or cardiovascular disease induced by ETS. There is no hard data to use as an educational tool to induce concern in the nonsmoker. No hard data to induce the concern in the smoker that he or she may be posing a threat to the health of workmates. Unreasonable as it is, it is being carried out by government and anti-smoking organisations in their campaigns.

I recall and bring to your attention the views of Professor Patrick Lawther (40) in London in 1964, referring to chemicals:

"It would seem, however, that contemporary concern about pollution, laudable though it may be, is often inversely proportional to its concentration, for now, in some quarters, there may be seen all too frequently almost pathologically intense, and often irresponsible, propaganda against pollution of any sort, despite the absence of incriminating evidence. It could be said in defence of such activity that any pollution must be bad, but we must first define clean air if we are not to finish by prohibiting any form of industrial or communal activity, including breathing. The currently common lack of a sense of perspective must be deplored because it can lead, through political pressure, to the installation of enormously expensive methods for abatement which can place wholly unjustifiable burdens on societies in which there are urgent needs for expenditure on unequivocal hazards to health."

Nonsmoking policies for workplaces are based on no scientific grounds, on extrapolations mainly from studies of spouses, and extrapolations from the epidemiology of smokers, faith in the no threshold theory for effects of toxic substance, an absence of an established no effect level, and possible plausibility. Such policies must therefore be considered as social ones and not health policies. Professor Weiss's (18) view I share. The use of passive smoking as a tactic in the smoking cessation strategy is based on weak grounds apart from irritations. I also share the concerns of the AFL/CIO (7).

The basic problem of ETS in the workplace, whether in the factory or office is that the risk, if it exists, is a small one and likely to be undetectible and confounded, however, a large population is exposed and uncertainty prevails. We are in the position described by Professor Rose (39) of the London School of Hygiene and Tropical Medicine, "In that state of uncertainty we have to avoid both the panic of the professional protester and the unfounded but seemingly unshakeable confidence of the professional experts".

Those involved in government anti-smoking activities should be aware of the tenuous nature of the data on passive smoking and health effects. The deliberate use of fear inducing tactics by misrepresenting such data by dubious extrapolations is propaganda and not health education.

## REFERENCES


1. White, J. and Froeb, H. (1980) New Engl. J. Med., 303, 393-394.
2. Kentner, M., Triebig, G. and Weltle, D. (1984) Prev. Med., 13, 656-659.
3. U.S. Surgeon General (1985) The health consequences of smoking: cancer and chronic lung disease in the workplace., U.S. Department of Health and Human Services, Rockville, Md.
4. Hirayama, T. (1981) Br. Med. J., 282, 183-185.
5. Trichopoulos, D., Kalandidi, A., Sparros, L. and Macmahon, B. (1981) Int.J. Cancer, 27, 1-4.
6. Garfinkel, L. (1981) J. Natl. Cancer Inst., 66, 1061-1066.
7. AFL-CIO (1985) Executive Council Statement on Smoking and the Workplace, Bal Harbour, Fl.
8. National Research Council (1986) Environmental Tobacco Smoke. Measuring Exposures and Assessing Health Effects, Washington, D.C.
9. U.S. Surgeon General (1986) The health consequences of involuntary smoking, U.S. Department of Health and Human Services, Rockville, Md.


2023383697



Source: https://www.industrydocuments.ucsf.edu/docs/zqyx0000

10. IARC Monographs on the evaluation of the carcinogenic risk of chemicals to humans, Vol 38, Tobacco Smoking (1986) Lyon, France.
11. Aviado, D. (1986) Smoking and Society, Ed. Tollison. pp.137-165.
12. Wald, N.J., Nanchahal, K., Thompson, S.G. and Cuckle, H.S. (1987) Brit. J. Med., 293, 1217-1222.
13. Lebowitz, M.D. (1986) Environ. Int., 12, 3-9.
14. Uberla, K. (1987) Int. Arch. Occup. Environ. Health, 59, 421-437.
15. Geneva (1983) ETS - Environmental Tobacco Smoke. Eds. R. Rylander, Y. Peterson and M.-C. Snella.
16. Vienna (1984) Symposium: Medical Perspectives on Passive Smoking, Preventive Medicine 13 (6).
17. Essen (1986) Proceedings of the International Experimental Toxicology Symposium on Passive Smoking, Ed. H. Mohtashamipur, Toxicology Letters, Amsterdam.
18. Weiss, S.T. and Speizer, F.E. (1987) (Editorial) Am. Rev. Resp. Diseases, 135, 1225-1226.
19. Hammond, E.C. and Selikoff, I.R. (1981) Environ. Res., 24, 444-452.
20. Bertram, J.F. and Rogers, A.W. (1981) Brit. J. Med., 283, 1567-1569.
21. Doll, R. and Peto, R. (1981) J. Natl. Cancer Inst., 66, 1193-1308.
22. Doll, R. (1978) Environ Health Perspect., 22, 23-31.
23. Curwen, M.P., Kennaway, E.L. and Kennaway, N.M. (1954) Brit. J. Cancer, 8, 181-198.
24. Haenszel, W. (1961) J. Nat. Cancer Inst., 26, 37-132.
25. Prindle, R. (1959) J. Air Pollut. Control Assoc., 9, 12-19.
26. Levin, M.L., Haenszel, W. Carroll, B.E., Gerhardt, P.R., Handy, V.H. and Ingraham, S.C. (1960) J. Natl. Cancer Inst., 24, 1243-1257.
27. Buell, P. and Dunn, J.E. (1967) Arch. Environ. Health, 15, 291-297.
28. Carnow, B.W. (1978) Environ. Health Perspect., 22, 17-21.
29. Dean, G. (1959) Brit. Med. J., 2, 852-857.
30. Dean, G. (1964) Proc. Roy. Soc. Med., 57, 984-987.
31. Eastcott, D.F. (1956) Lancet, 1, 37-39.
32. Robertson, A.S., Burge, P.S., Hedge, A. (1985) Br. Med. J., 291, 373-376.
33. Finnegan, M.J., Pickering, C.A.C. and Burge, P.S. (1984) Br. Med. J., 289, 1573-1575.
34. Melius, J., Wallingford, K. Keenleyside, R., et al. (1984) Meeting of the American Congress of Government Hygienists.
35. Health and Welfare Medical Services (1986) Cited by Sterling.
36. Sterling, T.D., Collett, C.W., and Sterling, E.M. (1987) J. Occup. Med., 29, 57-62.
37. Kodama, A.M. and McGee, R.I. (1986) Arch. Environ. Health, 41, 306-311.
38. Rose, G. (1987) Br. Med. J., 294, 963-965.
39. Pearce, N.E. and Howard, J.E. (1986) Int. J. Epidemiol., 15, 456-462.
40. Lawther, P.J. (1985) The chemical industry and the health of the community, Ed. F.J.C.Roe. pp.25-27.

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# SMALL-AIRWAYS DYSFUNCTION IN NONSMOKERS CHRONICALLY EXPOSED TO TOBACCO SMOKE

JAMES R. WHITE, PH.D., AND HERMAN F. FROES, M.D.

**Abstract** We evaluated the effect of long-term passive smoking (involuntary inhalation of tobacco smoke by nonsmokers) and long-term voluntary smoking on specific indexes of pulmonary function in 2100 middle-aged subjects. Regardless of sex, nonsmokers chronically exposed to tobacco smoke had a lower forced mid-expiratory flow rate (FEF 25 to 75 per cent) and forced end-expiratory flow rate (FEF 75 to 85 per cent) than nonsmokers not exposed ($P<0.005$). In addition, values in passive smokers were not significantly different from those in light smokers and smokers who did not inhale ($P<0.005$). When we looked at the extent to which smoke exposure is related to graded abnormality, we found that nonsmokers in smoke-free working environments have the highest scores on the spirometric tests; passive smokers, smokers who do not inhale, and light smokers score similarly and significantly lower; and heavy smokers score the lowest ($P<0.005$). We conclude that chronic exposure to tobacco smoke in the work environment is deleterious to the nonsmoker and significantly reduces small-airways function. (N Engl J Med. 1980; 302: 720-3.)

IT is generally believed that infrequent and short-term exposure to pollutants in tobacco, such as carbon monoxide, nicotine, benzo(a)pyrene, and oxides of nitrogen, will not permanently alter pulmonary function in healthy adult nonsmokers. We tested two hypotheses: that nonsmokers chronically exposed at work to the pollutants in tobacco will score lower on tests of small-airways function than nonsmokers not chronically exposed to tobacco smoke; and that exposure to tobacco smoke will cause a graded abnormality in small-airways function in relation to the extent of smoke exposure.

## METHODS

### Subjects

To examine these hypotheses we used the scores on tests of mean forced mid-expiratory flow (FEF 25 to 75 per cent) and mean forced end-expiratory flow (FEF 75 to 85 per cent); these tests have proved effective in detecting small-airways disease in its early stages.[1-3] Forced vital capacity and forced expiratory volume in one second ($FEV_1$) were also studied.

Data were collected on 5210 cigarette smokers and nonsmokers who had been physiologically evaluated during a "Physical Fitness Profile" course sponsored by the Department of Physical Education, University of California, San Diego, between 1969 and 1979. Most of the subjects resided in San Diego, an area low in air pollution. Occupation and locations of work and residence according to zip codes were analyzed. There were no statistically significant differences between the groups in types of occupation or in working or living locations. Eighty-three per cent of the working subjects held professional, managerial, or technical positions, and the remainder were blue-collar workers. Personal habits, environmental pollution, and smoking habits were assessed from an extensive, self-administered questionnaire completed on two separate occasions. The reliability coefficients for the test and retest are greater than $r = 0.96$. Each subject's age, height, and weight were recorded, and they were categorized according to sex, exposure to tobacco smoke, and smoking habits.

From the Department of Physical Education, University of California, San Diego (address reprint requests to Dr. White at the Department of Physical Education, C-017, University of California, La Jolla, CA 92037).

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From the original 5210 candidates, 2208 were disqualified because they indicated on the questionnaire that they had a history of pulmonary or cardiac disease, persistent cough, recent asthma, respiratory illness, or bronchial disturbances; that they had had occupational exposure to dust or other toxic fumes; that they had lived in a smoggy or industrial area; or that they had been employed in areas associated with industrial pollution. The 3002 remaining candidates were assigned to one of six groups according to their exposure to tobacco smoke (Table 1). From each group 200 men and 200 women were then randomly selected and assigned to the comparison groups. However, only 50 male and female subjects were available for the noninhaling smoking group (3), and so the total number of subjects reported on is 2100.

A single technician administered successive forced-vital-capacity maneuvers until reproducible curves were obtained on each subject with use of the Donti Pulmonary Performance Analyzer (PA70). The fast vital-capacity spirogram achieving the greatest volume was used to calculate the forced vital capacity (FVC), the forced expiratory flow for one second ($FEV_1$), the forced mid-expiratory flow (FEF 25 to 75 per cent), and the forced end-expiratory flow (FEF 75 to 85 per cent) (Table 1).

Comparisons were made between the scores achieved by six groups of subjects matched for age and sex. Group 1 comprised nonsmokers who had neither lived in a house where tobacco smoking was permitted nor been employed in an enclosed working area that permitted smoking or routinely contained tobacco smoke. Group 2 comprised passive smokers, that is, nonsmokers who lived in a house where tobacco smoking was not permitted but had been employed for 20 years or more in an enclosed working area that permitted smoking and routinely contained tobacco smoke. Group 3 contained pipe, cigar, or cigarette smokers who did not inhale; Group 4, light smokers who had inhaled one to 10 cigarettes per day for 20 years or more, Group 5, moderate smokers who had inhaled 11 to 39 cigarettes per day for 20 years or more; and Group 6, heavy smokers who had inhaled more than 40 cigarettes per day for 20 years or more. Presumably, the subjects in these last four groups all worked in environments where smoking was permitted, since they themselves smoked at work.

To test the hypothesis that there was no difference between the pulmonary scores in each group, we used a statistical package called "SPSSH Release 6.02," which gave us a one-way analysis of variance. When the analysis of variance revealed a significant difference, the hypothesis was rejected. We then used the Student-Newman-Keuls multiple-comparison test to determine subgroups.[4]

### Carbon Monoxide Levels in Working Environment

On the questionnaire the subjects who did not smoke indicated whether smoking was permitted in their working area and whether the air generally contained tobacco smoke. To obtain a more objective measure of true concentrations of smoke in the working areas, we placed a portable carbon monoxide analyzer (Ecolyzer, Ener-

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Table 1. Vital Capacities and Expiratory Flow Rate (Mean ±S.D.) in Male and Female Smokers and Nonsmokers.

| Group | No. of Subjects | Sex | Age | Height | FVC | | $FEV_1$ | | FEF 25–75% | | FEF 75–85% | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | yr | cm | liters | % predicted | liters | % predicted | liter/sec | % predicted | liter/sec | % predicted |
| 1 — Nonsmokers, no smoky environment | 200 | F | 47.77±7.0 | 162.05±6.58 | 3.35±0.63 | 102 | 2.63±0.63 | 104 | 3.17±0.74 | 108 | 1.03±0.38 | 112 |
| | 200 | M | 48.8±7.59 | 176.3±7.77 | 4.91±0.75 | 102 | 3.72±0.65 | 103 | 3.78±0.79 | 104 | 1.22±0.35 | 120 |
| 2 — Nonsmokers, smoky environment > 20 yr | 200 | F | 45.91±6.94 | 161.8±6.68 | 3.23±0.46 | 98 | 2.47±0.63 | 99 | 2.72±0.71* | 93 | 0.78±0.36* | 85 |
| | 200 | M | 49.1±7.61 | 176.02±6.55 | 4.78±0.77 | 99 | 3.54±0.61 | 98 | 3.30±0.77* | 91 | 0.97±0.34* | 95 |
| 3—Smokers not inhaling cigarettes, pipe, or cigars > 20 yr | 50 | F | 46.93±7.10 | 159±7.09 | 3.19±0.52 | 97 | 2.49±0.74 | 99 | 2.71±0.87* | 92 | 0.78±0.43* | 85 |
| | 50 | M | 47.6±7.38 | 174.5±7.42 | 4.63±0.86 | 96 | 3.56±0.76 | 99 | 3.32±0.86* | 92 | 0.89±0.47 | 87 |
| 4 — Smokers inhaling 1–10 cigarettes per day > 20 yr | 200 | F | 47.36±7.0 | 159.77±7.44 | 3.15±0.39 | 96 | 2.40±0.62 | 98 | 2.63±0.73* | 89 | 0.76±0.31* | 83 |
| | 200 | M | 48.5±7.51 | 175.9±7.44 | 4.58±0.77* | 95 | 3.49±0.62* | 97 | 3.23±0.78* | 89 | 0.79±0.36* | 77 |
| 5 — Smokers inhaling 11–39 cigarettes per day > 20 yr | 200 | F | 45.72±6.87 | 160±6.99 | 2.80±0.38* | 85 | 2.13±0.62* | 85 | 2.29±0.70* | 78 | 0.63±0.31* | 69 |
| | 200 | M | 48.3±7.49 | 176.02±7.67 | 4.04±0.74* | 84 | 3.08±0.61* | 86 | 2.73±0.81* | 76 | 0.69±0.29 | 68 |
| 6 — Smokers inhaling > 40 cigarettes per day > 20 yr | 200 | F | 45.98±6.73 | 159.26±7.29 | 2.55±0.38* | 78 | 2.01±0.64* | 80 | 2.12±0.72* | 72 | 0.57±0.33* | 62 |
| | 200 | M | 47.8±7.44 | 176.53±7.9 | 3.92±0.73* | 82 | 2.77±0.60* | 77 | 2.59±0.82* | 72 | 0.61±0.31* | 60 |
| Prediction for age: 46.8 yr, height 160.53 cm † | 1050 | F | 46.61±6.94 | 160.3±6.99 | 3.28 | — | 2.52 | — | 2.94 | — | 0.92 | — |
| Prediction for age: 48.3 yr, height 175.77 cm † | 1050 | M | 48.35±7.50 | 175.77±7.54 | 4.81 | — | 3.60 | — | 3.62 | — | 1.02 | — |

*Significantly different from values in nonsmokers ($P > 0.005$). †Predicted according to Morris.[11]

getics Science, Elmsdorf, N.Y.) on top of the desk or in the working area of 40 randomly selected nonsmokers who had indicated that they worked in an environment without smoke and 40 similarly selected nonsmokers who had indicated that the air in their working area contained smoke from co-workers. Carbon monoxide, a component of tobacco smoke, is an accurate tobacco-smoke tracer,[5-7] and its concentration is directly proportional to that of tobacco smoke.[8,9] The mean carbon monoxide concentrations in areas where smoking occurred were compared with similar measurements taken in areas where smoking was not permitted (Table 2). During the day, particularly at 11:20 a.m. and 1:20 p.m., the differences in the mean values between environments with and without smoking were significantly different. Only at 7:00 a.m. and 7:00 p.m., before and after working hours, were carbon monoxide concentrations the same. In the environments where there was smoking, the mean concentration at peak values almost doubled, from 6.4 to 11.6 parts per million, whereas in the environments where there was no smoking, the increase was from 6.3 to 8.2 parts per million. The peak concentration of carbon monoxide was significantly greater in the environments where smoking occurred. The carbon monoxide analyzer was calibrated in the laboratory and found to have ±2 per cent reproducibility and ±2 per cent accuracy at levels between 0 and 50 parts per million.

Of the 80 working areas tested for carbon monoxide concentrations, 76 were air-conditioned. No attempt was made to determine the direction of the circulation of refrigerated air or the exact air exchange. Building codes require a minimum of five to six complete air exchanges per hour. The size of the rooms and the number of coworkers in the working areas were computed. In terms of these two factors, there was no significant difference between the working areas where smoking was permitted and where it was not permitted. In the areas without smoking, ventilation was sufficient to maintain peak carbon monoxide concentrations below 9 parts per million, which Holbrook defines as the upper limit for carbon monoxide in areas with adequate ventilation.[10] However, ventilation in our study was not capable of adequately extracting polluted air as measured by carbon monoxide levels in areas where smoking was allowed (Table 2).

## Results

The mean values ± the standard deviation for the FVC, $FEV_1$, FEF 25 to 75 per cent, and FEF 75 to 85 per cent in the six groups studied are shown in Table 1. There were no statistical differences in the ages and heights within the various groups.

Compared with nonsmokers who worked in environments where there was no smoking, both the men and the women in the other five groups had significantly lower values for FEF 25 to 75 per cent and FEF 75 to 85 per cent. These lower levels were observed in both absolute values and per cent predicted values calculated according to the formulas of Morris.[11] The FVC and $FEV_1$ were not as sensitive, and values were lower only in the female heavier smokers of Groups 5 and 6 and in the male heavier smokers of Groups 4, 5,

Table 2. Carbon Monoxide Concentrations (parts per million) Measured during the Workday.

| Time | Smoking Permitted in Work Area (40 Subjects) | | Smoking Not Permitted in Work Area (40 Subjects) | | Comparison between Working Areas |
|---|---|---|---|---|---|
| | mean ±S.D. | Range | mean ±S.D. | Range | T value |
| 7:00 a.m. | 6.4±2.9 | 3.1–10.9 | 6.3±1.7 | 3.3–10.3 | 0.1 |
| 10:00 a.m. | 9.2±5.4 | 3.5–29.4 | 7.1±3.3 | 3.6–13.6 | 2.1* |
| 11:50 a.m. | 11.1±6.0 | 4.1–21.2 | 8.2±4.1 | 3.8–12.7 | 2.9† |
| 1:20 p.m. | 11.6±7.3 | 3.8–25.8 | 6.9±2.7 | 4.0–13.8 | 4.7† |
| 4:20 p.m. | 9.7±5.9 | 3.3–20.2 | 7.5±3.8 | 3.3–11.9 | 2.2* |
| 7:00 p.m. | 7.1±3.3 | 3.2–14.7 | 6.5±2.2 | 3.4–10.7 | 0.6 |

*$P = 0.05$ by unpaired t-test. †$P = 0.01$ by unpaired t-test.

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and 6. The passive smokers not only scored significantly lower than their nonsmoking counterparts but also fell into the same state of impaired performance as the noninhalers and light smokers. Again, this was true for both men and women.

Table 1 shows the predicted values for the average man and woman according to height and weight. There is no statistical difference between these predicted values and the values in nonsmokers. However, as the degree of smoking exposure increased in both men and women, the performance of FEF 25 to 75 per cent and FEF 75 to 85 per cent decreased, as shown by the stepwise reduction in scores for moderate and heavy smokers (Table 1).

As shown in Table 3, differences in pulmonary performance between groups were analyzed by statistical analyses of variance according to the Student-Newman-Keuls multiple-range test at a level of 0.005.[4] The subgrouping indicates that both male and female nonsmokers who live and work in a smoke-free environment score the highest of all the subgroups, and that passive smokers, smokers who do not inhale, and light smokers are not significantly different from one another. The analysis also shows that both moderate and heavy smokers are, in general, not significantly different: that is, they share about the same degree of dysfunction. It is impressive that in all pulmonary-function tests, the moderate and heavy smokers scored significantly worse than all other groups, and that in tests that best reflect small-airways function (FEF 25 to 75 per cent and FEF 75 to 85 per cent), the nonsmokers scored significantly better than all other groups.

Table 3. Pulmonary Function in Subjects Chronically Exposed to Tobacco Smoke.*

| Variable | Sex | Subgroups Arranged in Order of Decreasing Pulmonary Function |
|---|---|---|
| FVC | Female | NS NI LS PS MS HS |
| | Male | NS PS NI LS MS HS |
| $FEV_1$ | Female | NS NI LS PS MS HS |
| | Male | NS NI PS LS MS HS |
| FEF 25–75% | Female | NS PS NI LS MS HS |
| | Male | NS NI PS LS MS HS |
| FEF 75–85% | Female | NS PS NI LS MS HS |
| | Male | NS NI PS LS MS HS |

*Underlinings indicate subgroups arranged by the Student-Newman-Keuls multiple-range test at the 0.005 level. For example, for FVC, female, there are three groups that differ from each other statistically; the first group comprises NS, NI, LS, and PS, the second group MS, and the third HS. NS denotes nonsmokers, PS passive smokers, NI noninhaling smokers, LS light smokers (one to 10 cigarettes per day), MS moderate smokers (11 to 39 cigarettes per day), and HS heavy smokers (40 or more cigarettes per day) — Groups 1 to 6, respectively.

## Discussion

We used several methods to minimize the standard error of the difference and eliminate sampling biases in this study. First of all, measurements were made in a large number of subjects (a total of 2100), and they were divided into six specific groups according to their responses to a questionnaire on smoking history. Candidates with health, environmental, or occupational conditions that could influence pulmonary function adversely were disqualified from the study. Furthermore, comparisons among the groups of occupations and working and living locations showed that they were not significantly different, thus minimizing the sampling error. When the sites for measurement of carbon monoxide were selected, bias was reduced because 20 male and 20 female nonsmokers and 20 male and 20 female passive smokers were randomly selected from the 200 subjects in each group. Finally, most studies on smoking have a correlational design; this approach may weaken many of the conclusions because tobacco smoking is a matter of choice and is done for a variety of personal reasons, which may cause both the smoking and the pulmonary dysfunction. In our study, neither the nonsmokers nor the passive smokers chose to smoke; therefore, the pulmonary dysfunction found in passive smokers cannot be attributed to the "reasons" that may be related to the dysfunction in smokers. Comparison between the nonsmokers and the passive smokers is thus truly experimental.

Although there was no statistical difference between the predicted values in Table 1 and values in the nonsmokers, it must be remembered that predicted values were based on the combination of data obtained from nonsmokers and passive smokers. When data on the nonsmoker and the passive smoker are arithmetically averaged, the value approximates that found in existing predicted norms. We believe that in choosing subjects for establishing "normal predicted values," one should take into account the degree of cigarette pollution in which the subjects live and work.

Ambient carbon monoxide may have deleterious effects on bodily functions other than those of the lung. Studies by Bridge and Corn[9] and by Hexter and Goldsmith[12] have indicated that concentrations of carbon monoxide as low as 8 parts per million can increase the incidence of symptomatic or overt ischemic heart disease. It has also been shown that elevated carbon monoxide concentrations can increase the incidence of early angina in patients with atherosclerotic heart disease.[13,14] High carbon monoxide concentrations also lead to alterations in psychomotor performance in healthy subjects.[15] We chose to look at long-range changes in the function of small airways in the lungs of nonsmokers chronically exposed to low levels of tobacco smoke as measured by carbon monoxide levels. Carbon monoxide was used as an index of exposure to

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tobacco smoke and was not intended to be identified as the specific inciting agent.

The traditional spirometric tests of FVC and $FEV_1$ depend mostly on the total airway resistance and elastic recoil of the lung and are often normal in the presence of extensive small-airways disease.[16] In contrast, the mid-expiratory and maximal end-expiratory flow rates reflect expiratory flow in the presence of lower lung volumes during a period when airway segments may be in the process of closing.[17,18] Other studies have indicated a high frequency of small-airways disease in relatively asymptomatic cigarette smokers.[19] In one study, 72 per cent of the subjects had significantly decreased maximal mid-expiratory flow rates.[1] Morris[2,20] has shown that measurements of forced expiratory flow rate are simple and accurate predictors of changes in small airways. It is generally agreed that in the presence of normal FVC and $FEV_1$, reduced forced mid-expiratory and end-expiratory flow rates are commensurate with small-airways disease.[2,11,20] Our results agree with those of Macklem and Mead[16] and of Morris,[2,11,20] in that neither FVC nor $FEV_1$ was significantly different in passive smokers and nonsmokers, but that both FEF 25 to 75 per cent and FEF 75 to 85 per cent were significantly lower in passive smokers than in nonsmokers.

In considering the relation of graded abnormality to the extent of smoke exposure, it is interesting to note that the nonsmokers in our study scored well above all other groups in the tests in Tables 1 and 3. However, there was no significant difference in the scores of the passive smokers, the smokers who did not inhale, and the light smokers. This finding suggests that if long-term small-airways dysfunction is occurring, the nonsmokers who work in a smoky environment have about the same risk of impairment as do smokers who do not inhale and smokers who inhale between one and 10 cigarettes per day. Niewoehner et al.[21] showed that further increases in exposure to cigarette smoke cause a progression from small-airways involvement to extensive bronchial and alveolar disease: the greater the exposure, the greater the involvement.

There is supporting evidence of the effects of passive smoking on small-airways function and the development of a graded abnormality according to extent of exposure to smoke. A recent study by Tager et al.[22] has shown that children living in households where parents smoked tobacco had lower mid-expiratory flow rates (FEF 25 to 75 per cent) than children who lived in households where smoking did not occur. In addition, FEF 25 to 75 per cent in children who had never smoked declined as a function of the number of parents who smoked in the household. The greater the exposure, the lower the pulmonary-function score.

Although many nonsmokers believe that exposure to tobacco smoke is irritating and generally obnoxious, our studies and Tager's show the adverse effects of passive smoking on the small-airways function of both adults and children. With these data now available, health officials and the medical profession must consider potential for small-airways dysfunction in nonsmokers chronically exposed to tobacco smoke.

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## References


1. McFadden ER Jr, Linden DA. A reduction in maximum mid-expiratory flow rate: a spirographic manifestation of small airway disease. Am J Med. 1972; 52:725-37.
2. Morris JF, Koski A, Breese JD. Normal values and evaluation of forced end-expiratory flow. Am Rev Respir Dis. 1975; 111:755-62.
3. Macklem PT, Thurlbeck WM, Frazer RG. Chronic obstructive disease of small airways. Ann Intern Med. 1971; 74:167-77.
4. Nie N, Hull C, Jenkins J, Steinbrunner K, Bent D. Statistical package for the social sciences. 2nd ed. New York: McGraw-Hill, 1975.
5. Huber GL. Smoking and nonsmokers — what is the issue? N Engl J Med. 1975; 292:858-9.
6. Stewart RD. The effects of low concentrations of carbon monoxide in man. Scand J Respir Dis [Suppl]. 1974; 91:56-62.
7. Cohen SI, Perkins NM, Ury HK, Goldsmith JR. Carbon monoxide uptake in cigarette smoking. Arch Environ Health. 1971; 22:55-60.
8. Hoegg UR. Cigarette smoke in closed spaces. Environ Health Perspect. 1972; 2:117-28.
9. Bridge DP, Corn M. Contribution to the assessment of exposure of nonsmokers to air pollution from cigarette and cigar smoke in occupied spaces. Environ Res. 1972; 5:192-209.
10. Holbrook JH. Tobacco and health. CA. 1977; 27:344-53.
11. Morris JF. Spirometry in the evaluation of pulmonary function. West J Med. 1976; 125:110-8.
12. Hexter AC, Goldsmith JR. Carbon monoxide: association of community air pollution with mortality. Science. 1971; 172:265-6.
13. Aronow WS, Harris CN, Isbell MW, Rokaw SN. Effect of freeway travel on angina pectoris. Ann Intern Med. 1972; 77:669-76.
14. Aronow WS, Isbell MW. Carbon monoxide effect on exercise-induced angina pectoris. Ann Intern Med. 1973; 79:392-5.
15. Mikulka P, O'Donnell R, Heinig P, Theodore J. The effects of carbon monoxide on human performance. Ann NY Acad Sci. 1970; 174:301-12.
16. Macklem PT, Mead J. Resistance of central and peripheral airways measured by a retrograde catheter. J Appl Physiol. 1967; 22:395-401.
17. West JB. Respiratory physiology — the essentials. Baltimore: Williams & Wilkins, 1974.
18. *Idem.* Pulmonary pathophysiology — the essentials. Baltimore: Williams & Wilkins, 1977.
19. McCarthy DS, Spencer R, Greene R, Milic-Emili J. Measurement of "closing volume" as a simple and sensitive test for early detection of small airways disease. Am J Med. 1972; 52:747-53.
20. Morris JF. Chronic obstructive pulmonary disease. 5th ed. New York: American Lung Association, 1977.
21. Niewoehner DE, Kleinerman J, Rice DB. Pathologic changes in the peripheral airways of young cigarette smokers. N Engl J Med. 1974; 291:755-8.
22. Tager IB, Weiss ST, Rosner B, Speizer FE. Effect of parental cigarette smoking on the pulmonary function of children. Am J Epidemiol. 1979; 110:15-26.


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this place from the inside, in the way that those of us who serve here do.

The press and media people pack the gallery upstairs during a debate on a pay raise, but they rarely write or speak about the personal ramifications of the job here. This is not to complain. Every Member here feels that it is a supreme honor to serve as a Member.

But it is important that the public know more about what kinds of people serve here, how hard they work, the personal toll that is taken on individuals and families.

I have seen grown men here cry when their marriages were destroyed. I have seen grown men depressed about missing events that were important to their children.

In sports they have a term called "playing hurt," when players participate even when injured. Well, I have seen Members of the House "play hurt." Members who were ill and dragged themselves to the airport to keep an important commitment back home.

I have seen Members with serious illness or disease struggle to do a good job, to make rollcall votes and committee meetings.

The picture of the Congress presented to the public via the Abscam scandal was not a fair one. This is not a place where you can reach in and pick any seven Members and expect that they will accept money in a brown paper bag. It is not that kind of place and it is an insult to all who have served well here that such a picture of the Congress be presented.

Scandal is what sells newspapers and soap on the evening news. Pay raises and debates over them in this body are in the same category. But these things do not begin to tell the story of what happens here on a day-to-day basis.

What happens here on a day-to-day basis is a struggle to get things done, to do the right thing for our districts, for the Nation and the world. What gets in the way of that is familiar to all of us.

The hectic schedule is a problem. The lack of predictability in the scheduling. The tug and pull of various people and interests wanting our attention. The seemingly constant attention to getting reelected. The accompanying preoccupation with raising money. And, of course, the impact that such a preoccupation has on policymaking.

There must be a movement developed to deal with these problems. I believe we should move to a 4-year term with a limit of three terms in the House and two 2-year terms in the Senate. I believe we should move to a 2-year budget and plug in a requirement that the Congress do meaningful oversight for 3 or 4 straight months each year.

Most importantly, we need campaign reform—a limit on PAC's, public financing, free access to media, and many other important changes. The political system is being contaminated by money and we must do something about it.

Such dramatic changes must take place with pressure from the outside and I intend to do my share as a private citizen in that regard.

Being a private citizen again will not be altogether unhealthy. Getting out of politics for a time will give me an important perspective.

But there will never be anything to take the place of the warm friendships I have developed here. I have not had the chance to thank each of my colleagues and staff for their kindness. I hope this statement will at least partially serve that purpose.●

## WHITE-FROEB STUDY DISCREDITED BY SCIENTISTS

**HON. L. H. FOUNTAIN**
OF NORTH CAROLINA
IN THE HOUSE OF REPRESENTATIVES

*Thursday, December 16, 1982*

● Mr. FOUNTAIN. Mr. Speaker, after 30 years of service to the people of the Second District of North Carolina, I am about to retire from the U.S. House of Representatives. Before leaving I would like to submit, for the RECORD, an item dealing with an issue with which I and many others have long been interested, namely, the alleged effect of smoking on the health of the nonsmoker.

Mr. Speaker, let me briefly place the issue into its proper context. In 1978, the Subcommittee on Tobacco of the House Committee on Agriculture heard testimony from a vast array of eminent scientists and physicians on the issue of the effect of tobacco smoke on nonsmokers. Those individuals who testified generally agreed that no conclusive scientific evidence exists to support the claim that smoking affects the health of nonsmokers. In 1980, however, an article appeared in the New England Journal of Medicine by Drs. White and Froeb entitled "Small Airways Dysfunction in Nonsmokers Chronically Exposed to Tobacco Smoke," in which the authors concluded that smoking in the workplace adversely affects the lung function of nonsmokers. This conclusion appeared to conflict with the testimony presented to the Subcommittee on Tobacco.

Since its publication, the White-Froeb study has been used to support both regulatory and legislative activities in the United States. For example, the study was referred to in testimony before the Civil Aeronautics Board during its recent consideration of rules regarding smoking aboard commercial aircraft. The National Research Council report entitled "Indoor Pollutants" which was issued in 1981 under an EPA contract also relies on the study. Finally, the White-Froeb study has received widespread attention in both State and local legislative and policymaking bodies.

The White-Froeb study continues to play an important role in legislative considerations, despite the fact that the study itself has been heavily criticized by scientists and health practitioners. Most recently, at the 1982 joint meeting of the American Lung Association-American Thoracic Society, Dr. Michael D. Lebowitz, professor of internal medicine, college of medicine, University of Arizona and special consultant to the Subcommittee on Tobacco, presented reasons why, in his own words, "the results of this study cannot be used to demonstrate an effect of passive smoking on forced expiratory flows in adults exposed in the workplace." Dr. Lebowitz, a noted specialist in epidemiology and respiratory diseases, said that the basic problem with the White-Froeb study is that it is "improperly designed" and that "there are problems with the whole data set and with the conclusion." Dr. Lebowitz also expressed concern that the significance of the White-Froeb data appeared to depend upon their unexplained omission of data from 3,000 subjects originally included in the study.

Mr. Speaker, Dr. Lebowitz wrote a letter, dated July 10, 1981, to our colleague, Congressman CHARLES ROSE, Chairman of the Tobacco and Peanuts Subcommittee of the House Agriculture Committee, as a result of a personal interview which Chairman ROSE and Dr. Lebowitz had with Dr. White. With the personal consent of Chairman ROSE, I am inserting herewith Dr. Lebowitz's letter. It more fully explains the author's views regarding the White-Froeb study.

I also want to mention another evaluation of the White-Froeb study, one which was made by Dr. J. G. Gostomzyk, director of the department of health of the city of Augsburg, West Germany. After an extensive, detailed review of the White-Froeb study, Dr. Gostomzyk has concluded that the White-Froeb data were incompletely presented and did not satisfy the prerequisites for scientific credibility. In addition, Dr. Gostomzyk remarked that "Dr. White's methodology is not scientific but that of a lay person with convictions," and concluded that "we assume that Dr. White's study is an attempt at scientific validation of his credo and that he possibly is unaware of the inadequacy of this methodology." It is obvious that Dr. Gostomzyk is referring to Dr. White's outspoken antismoking activities in California, including Dr. White's endorsement of public smoking referendums which were, incidentally, twice rejected by the California voters.

Given these and other criticisms of the White-Froeb study, it would appear that the New England Journal of Medicine has, perhaps unwittingly, performed a disservice to its readership. It is extremely unfortunate that a study so fraught with methodological problems, as indicated through

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numerous criticisms by scientists in the United States and elsewhere, should have been published in such a reputable journal of medicine. The White-Froeb study should, therefore, not be relied upon by the Congress, Federal agencies, or other legislative or policymaking bodies when considering restrictions on smoking in public places.

THE UNIVERSITY OF ARIZONA,
COLLEGE OF MEDICINE,
*Tucson, Ariz., July 19, 1981*

Congressman CHARLES ROSE,
*Chairman, Subcommittee on Tobacco and Peanuts, House of Representatives, Rayburn Building, Washington, D.C.*

DEAR CONGRESSMAN ROSE: The following is a summary of my notes on our visit to Dr. James White at UC San Diego, as per our discussion. Unfortunately, despite the statement in the editorial of the New England Journal of Medicine (27 March 1980), Dr. White and his co-author did not "faultlessly demonstrate a reduction in measures of small airways of healthy non-smokers exposed to cigarette smoke in the work place". It is apparent from our visit and the article that there were various faults in the present study, which shall be discussed.

The problems with the research design are as follows:

The participants were not only volunteers, but generally had to pay for the physical fitness course; this is the reason most were white-collar. Employees in specific factories invited White to run the physical fitness course in their factories as well, which would also bias the population sample. Blue-collar workers were not distributed randomly. (It has to be assumed that volunteers in the physical fitness courses fall into unrepresentative categories: the highly motivated, with an interest in health and usually healthier, those who are worried about health and generally less healthy; the first group would include fewer smokers and the second group would include more smokers.)

The questionnaire utilized was not a validated one per se; test-retest comparisons were made only on the smoking questions and very small groups of subjects. The smoking information was not validated. There were no test-retest or validations on symptoms asked in the questionnaire. The questionnaire itself was derived by the investigator, and included some questions from standard questionnaires; this did not appear to include standard respiratory questions, and in fact various typical respiratory questions (such as phlegm) were not asked. The questionnaire did not include questions on attitude, but did include questions on activity levels and jobs (duration, type). The questionnaire did ask how many smokers were in their work area, room size, and nature of the air conditioning. It also included questions about residences in the last 20 years (zip codes), so that exposures away from work were assessed by residential location. A question was asked about smokers in the home. (Thus, the smoking information is not validated, but is probably relatively accurate. The information about exposure to passive smoking is only approximate, as is the information on other occupational exposures. Exposures to air pollutants or to unknown toxic gases in the working place is only approximate, and their effects underestimated.)

Dr. White presented a paper to the American College of Sports Medicine, the abstract for which in 1977 indicated there were 7,122 subjects enrolled between 1969 and 1977; However, in the New England Journal of Medicine article, he states that the base population analyzed is only 5,210 smokers and non-smokers enrolled between 1969 and 1979. Although he excluded all the ex-smokers, some whose zip codes were missing, his answers as to why the rest of the subjects were excluded were entirely unclear and tend to indicate potential bias in selection of subjects for consideration for analyses. It might be added that the 2,100 subjects analyzed in the NEJM article and those analyzed and presented in the Sports Medicine abstract appear to be the same as they yield exactly the same table of results (as determined from comparison of the table in the Sports Medicine manuscript and the NEJM table).

In addition to the sources of bias mentioned above, it is apparent that the non-smokers in clean work environments and those in smoking work environments have not only chosen not to smoke, but it is likely that those non-smokers working in smoking environments may be different for a variety of reasons from non-smokers working in clean environments. Furthermore, it is apparent that the non-smokers in non-smoking environments are quite different in that their lung function is "super normal" in comparison even with the Seventh Day Adventists (the source of the Morris prediction equations).

Dr. White did state that from the questionnaire and from the baseline tests that there were no significant differences in the three non-smoking/non-inhaling groups in terms of the amount of previous exercise or oxygen consumption, but he was unsure of the difference in percent of body fat. Smokers did have less body fat, were less in terms of having lower oxygen consumption, and had less activity. He says further that there were no differences between the groups in terms of childhood respiratory history (lower respiratory tract illnesses) from his submitted questionnaire information, but he did not ask about family history. He did not ask sufficiently about respiratory questionnaires to appropriately exclude groups on the bases of productive cough ("cough bronchitis"). He states that there were no differences in prevalence rates of questionnaire responses by zip codes; if so, this contradicts other evidence vis-a-vis the effects of air pollution in these areas. He was not able to assess other exposures such as those from hobbies, exposures to gas stoves, or transportation. In terms of passive smoking in the home, he excluded such passive smokers from the non-smoking and passive smoking groups, but not from any smoking groups. He was not able to provide any information about the distribution of characteristics in those eliminated from the original 7,000 or the 2,208 that qualified because of other questionnaire results.

With regards to the pulmonary function testing done by Dr. White, it must be first noted that the instrument used is not considered a satisfactory instrument in that it is non-linear (highly biased) at both high volumes and low volumes. (This has the effect of maximizing differences in that anyone with minor aberrations of total vital capacity or of flows at the end of the flow volume curve would have very different, that is, low, flows.) The comparisons that Dr. White did and reported on in his response letter in the NEJM (14 August 1980) would not in any way modify this opinion. Furthermore, Dr. White has the only pulmonary function technician and reader. Even though he was trained at the VA hospital and his techniques were evaluated by test—retest and by comparison to other readers, any biases inherent in Dr. White's thinking (see below) would affect the way he read the tests. Furthermore, he took the FEV, and flows off the same spirogram using an approximation technique published by Morris, et al., which is not an adequate or accurate representation of those measures. All of his tests were baseline tests done after two and a half hours in the classroom in the evening on those without acute respiratory illnesses (usually on a Monday or Tuesday evening); thus, there is probably little diurnal variation or pretest biases other than those experienced by the workers during their work day and in their activities prior to the classroom. Although it is difficult to judge the effects of these factors, they may have influenced the test results, especially in those with any significant exposures during the day.

The major problem with the pulmonary function test results as reported is that they are not age- and height-adjusted, since lung volumes and flow rates are associated with both of these factors. In other words, Dr. White used raw values of flows and volumes to do comparisons. He did this on the assumption that the mean age and height were similar for the different groups. This is a mistake, since the distributions for those ages and heights could have differed. Furthermore, his quoted figures for percent predicted are strictly for the average person, age 49, with an average height, and does not represent the group for which they are provided. In terms of these statistical analysis, he just chose the SNK package among many. There is no correlation coefficient per se. "Normality" was not an objective of this study, so he cannot state anything about the normality of the subjects studied, including those he considered to have significantly different results from the non-exposed non-smokers. He does not understand the difference between clinical meaningfulness and statistical significance. It is quite obvious that the majority of those in the passive smoking and in the non-inhaling group are quite normal and that very few would be considered abnormal by any criteria.

In Dr's reported results, he quotes as incorrect significance level of $p < .005$, whereas the level provided by the technique is $p < .05$. This is very different, given the number of comparisons made, and indicates that some of the results would not be significant if corrections were made for the number of comparisons. Furthermore, the data presented in Table 1 was used to recompute the SNK analysis by Mary C. Townsend, MPH (Department of Epidemiology, University of Pittsburgh). Those results differ from those published by Dr. White and are provided in the attachment. The most important of the differences is the finding that the passive smokers and light smokers differ for the male FEV 75-85 percent. Thus, the effect of passive smoking on non-smokers is still unconfirmed, despite Dr. White's unfailing conviction that it is confirmed.

Other minor points: In terms of the carbon monoxide sampling, although it is stated that it was randomized, it was really on only 40 smoking and 40 non-smoking situations chosen by chance but not by random selection. Dr. Froeb, the co-author with Dr. White, is a private practitioner in La Jolla and helped Dr. White in drafting the NEJM manuscript from the manuscript presented at the American College of Sport Medicine. It might be pointed out that San Diego is not strictly low in air pollution concentrations, nor uniformed throughout the area, this may bias some results. Dr. White performed the pulmonary function tests until "reproducible curves were obtained", but they do not necessarily follow the Intermountain, Snowbird, or ATS recommendations.

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In reviewing Dr. White's response to the letter to the Editor in the NEJM (14 August 1980), it is quite clear that Dr. White did not satisfactorily answer all the questions raised, many of which are similar to those raised in this letter. It is questionable, from the discussion, whether Dr. White would pursue any further re-analysis of the data, nor necessarily could it be pursued. It is questionable, given the basic underlying problems in the research design, that re-analysis of the data would be worthwhile. On the other hand, given other results that contradict Dr. White's, including those now in press (such as Comstock et al., Johns Hopkins, presented at the Society for Epidemiological Research in June of 1981), it would be likely that a panel discussion of passive smoking might be valuable. I will be glad to furnish further discussion or help in that matter.

Sincerely,

MICHAEL D. LEBOWITZ, Ph. D., F.C.C.P.,
*Professor of Internal Medicine.*●

## TOM BUTTERFIELD

## HON. IKE SKELTON

OF MISSOURI

IN THE HOUSE OF REPRESENTATIVES

*Thursday, December 16, 1982*

● Mr. SKELTON. Mr. Speaker, as 1982 draws to a close, it is customary to reflect upon the events of the past year. I would like to talk about an experience I had just over a year ago and about some sad news I heard just this week.

In December of 1981, a movie called, "The Children Nobody Wanted" was televised. This moving story depicted the work of a man named Tom Butterfield and the help he gave to fosterlings in Marshall, Mo. On Monday, December 13, my longtime friend, Tom Butterfield died of respiratory failure.

"The Children Nobody Wanted" is a true story. When Tom Butterfield was a freshman at Missouri Valley College in Marshall, Mo., he discovered the problems of children who have nowhere to go, and for whom the law makes few, if any, provisions. Boy by boy, he made a life for these homeless youngsters. Tom fought increasing odds, from the lack of money, to outdated laws. He became the youngest single adult—and the first bachelor—to be a legal foster parent in the State of Missouri. He and his boys rented an old country club and turned it into their ranch. Today, there are four ranches, giving a homelife to over 100 youngsters.

During this special time of the year, it is good to stop and think about the road we are traveling. Looking at the trail of Tom Butterfield's life, I can see that, although he died at the young age of 42, his contributions will go on for a very long time to come. It is appropriate, at this time of gift-giving, to look back at all the giving this man has done in his lifetime.●

## ADMINISTRATION'S INSENSITIVE APPROACH TO CANCER

## HON. ALBERT GORE, JR.

OF TENNESSEE

IN THE HOUSE OF REPRESENTATIVES

*Thursday, December 16, 1982*

● Mr. GORE. Mr. Speaker, the present administration appears to be headed down a regulatory path that will needlessly expose millions of people to many known, cancer-causing chemicals at levels well beyond those traditionally accepted as safe and prudent. One example, documented in hearings held before the Investigations and Oversight Subcommittee of the Committee on Science and Technology which I chaired, was EPA's failure to take rapid action in setting reasonable limits for exposure to formaldehyde, although it is unquestionably an animal carcinogen. My colleague, Hon. GEORGE E. BROWN, Jr. has done some excellent work in this area and has found similar evidence of EPA's failure to regulate certain pesticides that have been clearly shown to cause tumors in animals. Two recent articles in the New York Times (Dec. 4, 1982) and the Baltimore Sun (Dec. 8, 1982) provide further documentation of the new, high-risk approach to Federal cancer policy. I commend them to the attention of my colleagues. It is time for us to halt this administration's crass and insensitive bottom-line approach in which costs to industry are balanced against increased human suffering. We all owe a debt of gratitude to the gentleman from California and I am looking forward to his forthcoming subcommittee report on this subject.

The articles follow:

[From the Baltimore Sun, Dec. 8, 1982]

EPA AND CANCER: THE SHIFTING STANDARDS

(By Ken Cook)

WASHINGTON.—In what some critics charge is a fundamental and unjustified change in federal cancer policy, the Environmental Protection Agency (EPA) has determined that an insecticide which caused cancer in laboratory animals poses no cancer risk to human beings.

The decision removes the last barrier to the permanent registration of the insecticide permethrin for use on dozens of U.S. crops.

As a result of emergency exemptions granted by the agency since 1977, permethrin already is one of the country's major insecticides, used on millions of acres of vegetables, beans and cotton each year. Permethrin is marketed under the trade names Pounce and Ambush.

A leading critic of the EPA decision, Representative George E. Brown, Jr., (D., Calif.) characterizes the permethrin ruling as "one of several actions that suggest the EPA has adopted a new set of scientific principles in reaching regulatory decisions on proven animal carcinogens."

Federal pesticide law does not prohibit registration of cancer-causing chemicals if dietary and occupational exposure can be kept below the safety level established by the agency. By contrast, the Food and Drug Administration must by law prohibit the use of any food additive shown to cause cancer in laboratory animals.

The practical effect of a decision to brand permethrin as a human carcinogen would have been a greater restriction on the number of crops for which it could be registered. Such a ruling might also have left the manufacturers, FMC Incorporated of Philadelphia, and ICI, a British firm, more vulnerable to product liability suits.

John W. Melone, director of EPA's hazard evaluation division and author of the permethrin decision (which appeared in the *Federal Register* in October), said that one of the long-term animal studies submitted by a manufacturer in support of the chemical's registration "was clearly positive" for cancer, it reported that tumors had appeared on mice after they had been fed permethrin over an extended period. Mr. Melone said five other studies submitted by manufacturers were accepted by EPA as showing no cancer-causing effects in laboratory animals. However, Mr. Melone described one of those studies as "quite controversial" because EPA scientists could not agree on the results. A seventh study, also conducted by a manufacturer, was rejected because of irregularities in the way it was conducted.

Mr. Melone described the permethrin case as "unique" for having so many long-term studies available for scrutiny by EPA scientists. Only two such studies normally are required for pesticide registration.

"We concluded that the weight of evidence suggested this chemical is highly unlikely to be a potential human oncogen *(tumor-inducing substance)*," Mr. Melone said in a telephone interview. "And since we have to make a decision, we believe permethrin should not be regulated as a potential human oncogen."

Regarding the permethrin ruling, Mr. Melone said, "There's no question it's a change in perspective."

Representative Brown, chairman of the House Subcommittee on (Agriculture) Department Operations, Research and Foreign Agriculture, which has jurisdiction over EPA's pesticide program, said that "for the agency to adopt a policy which, in effect, attempts to balance positive tests with negative ones is clearly a bold step."

Mr. Brown considers the action "a monumental and quasi-scientific leap of regulatory faith."

There is a continuing debate among cancer experts over how to weigh various types of evidence to determine a chemical's potential to cause cancer in humans. Although the precise mechanisms of cancer remain unknown, scientists have for some time agreed that proven animal carcinogens do not all pose an equal risk of cancer to humans. Traditionally, cancer regulatory decisions, including those made by EPA, have been made as if they did.

In the past few years, several cancer researchers and research institutions have proposed detailed systems by which regulatory agencies might distinguish between "strong" and "weak" carcinogens, based on information gained from long-term animal studies such as those submitted in the permethrin case. Regulatory actions might then take the form of an outright ban if such a system ranked the chemical as a strong cancer risk. Chemicals judged to be weak would be regulated less stringently, or not at all.

Last summer, EPA circulated to a select group of experts a draft of a proposal to modify the agency's original cancer policy guidelines promulgated in 1976. The draft proposed a ranking system similar to one developed by the International Agency for Re-

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PREVENTIVE MEDICINE 13, 645–655 (1984)

# Influence of Passive Smoking on Pulmonary Function: A Survey[1,2]

MICHAEL D. LEBOWITZ

*Division of Respiratory Sciences, Westend Research Laboratories, University of Arizona Health Sciences Center, College of Medicine, Tucson, Arizona 85724*

A review of the effects of passive smoking on pulmonary function has been made. In children, there is still a great deal of confusion and controversy. The range of the many effects studied is limited; most prospective studies show small effects. In general, it appears that respiratory infections in children may be increased with passive smoking. The effects on children's pulmonary function ranges from 0 to 3% over the age range from conception to 20 years; these differences in absolute magnitude are considered small. In healthy adults, the effects on pulmonary function and symptoms are not considered to be of concern; the effects in asthmatics require further study. A study to evaluate the above effects is presented here. It derives from a prospective study of airway obstructive diseases in Tucson, Arizona. From that study, it was concluded that the effect of passive smoking on pulmonary function or respiratory symptoms (recorded on a daily basis) in children is not positive. Effects may be seen with regard to responses to other irritants in passive-smoking children. In adults, no effect was seen, even in those with asthma or airway obstructive diseases; this may be due to low dosage. It is concluded that further, more appropriate studies are needed to understand the role of passive smoking on pulmonary function. © 1984 Academic Press, Inc.

## INTRODUCTION

Although there is substantial agreement that passive, or involuntary, tobacco smoking produces annoyance and sensory irritant effects, its role in producing cardiovascular or respiratory diseases is less clear (34, 47). The specific contributions of passive smoke to personal exposures and their effects have not been sufficiently documented.

Several studies have shown that the levels of carbon monoxide and other sidestream smoke constituents in indoor areas are high in many instances (34, 43, 46, 47). Deposition of sidestream cigarette smoke in the human respiratory tract has been shown to be only about 11%, much less than mainstream smoke (16). The impact of environmental tobacco on indoor concentrations of nitrogen dioxide ($NO_2$) is usually small compared with the impact of gas stoves (64). Blood carbon monoxide (CO) levels may be higher in those exposed to passive smoke (36, 42), but are probably lower than the carboxyhemoglobin (COHb) levels associated with exposures to unvented gas stoves (34). Some of the same respiratory diseases may occur in indoor situations in which high levels of respirable suspended particulates (RSP) and gases are due to wood-burning or kerosene heaters (1, 53) and not to environmental tobacco smoke.

[1] This work was supported by NHLBI SCOR Grant HL14136.

[2] Presented at the Symposium "Medical Perspectives on Passive Smoking," April 9–12, 1984, Vienna, Austria.



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## PULMONARY FUNCTION IN ADULTS

There have been several studies of passive smoking and respiratory impairment in adults. Comstock *et al.* (6) evaluated respiratory symptoms and lung function in a community population and found some effects from gas stoves but no effects from passive smoking; no exposure measurements were made.

Lebowitz and his co-workers (21, 22) studied chronic respiratory symptoms and lung function in a community population and found no effects of passive smoking.

Kauffmann *et al.* (18) studied pulmonary function in adult populations in France (without indoor measurements or information about use of gas appliances). They found some effects of spouse's smoking on nonsmoking women, but the healthiest population lived in the most polluted areas. This indicated that the study might have been flawed by biased population selection, biased testing, or confounding factors. One epidemiological study of chronic bronchitis in Czechoslovakia (52) showed a significant increase of symptoms of this disease in passive smokers, but this occurred almost entirely among men and showed a much greater relation to other adverse factors and/or exposures.

The Harvard Six City Study (59) performed covariance analyses for adults, examining passive smoking, gas stove usage, and ambient pollution (by city) and controlling for age, sex, and smoking. It found no effect of passive smoking on the adult respiratory system. Other population studies reported at a 1983 NIH workshop (33) led to the conclusion that there was basically no effect on pulmonary function and maybe only slight, inconsistent effects on symptoms in adults.

White and Froeb (60) studied self-selected volunteers with regard to workplace exposure and reported some effects of passive smoking in a subset of their volunteers; their study has been discussed in detail (23). Even with a biased population, poor study design, and incorrect statistical evaluation, there were no clear-cut, consistent, medically meaningful differences between passive smokers and groups of nonsmokers; a corrected statistical analysis strengthened this conclusion.

Three physiological studies of the effects of environmental tobacco smoke exposure on healthy subjects have been conducted, and all have been negative with regard to such effects (39, 51), even when massive amounts of smoke were used to expose nonsmokers (41). Two similar studies of the effects of environmental smoking on asthmatics and controls have demonstrated conflicting findings. One study (50) showed no important effect on asthmatics or controls, whereas the other study (7) demonstrated a broncho-constrictor effect in highly reactive asthmatics at higher concentrations.

## PULMONARY FUNCTION IN CHILDREN

Studies of the relationship of children's lung function to parental smoking habits again show somewhat contradictory evidence. In some studies, such as those of Martinez *et al.* (29), Yarnell and St. Leger (63), and Kauffmann and Brille (17), effects of parental smoking were seen in terms of children's lung function; however, these did not measure or control for other indoor (or outdoor) pollutants.

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In other studies in which measurements were not made, contradictory results were found. Hasselblad *et al.* (15) demonstrated that maternal smoking habits explain 0.1% of the variance in children's forced expiratory volume (FEV) in the first 0.75 sec of the expiratory maneuver ($FEV_{0.75}$), but they utilized data from previous studies that had shown no such trend when examining other risk factors at the time (4, 14). Gas stove usage was not found to be significant in either study. Ekwo *et al.* (10) found respiratory effects on children from gas stove usage but inconsistent effects of passive smoking. Tager *et al.* (57) and Schenker *et al.* (48) found effects of parental smoking but not gas stoves; social status factors were again shown to be important.

Recently, several studies of passive smoking effects that included environmental monitoring have been performed. The results have been quite contradictory. In the studies by Florey *et al.* and Melia (11, 30, 31), looking at other indoor pollutants, they could find no relation of passive smoking to children's lung functions. Speizer *et al.* (55, 56) demonstrated the effects of gas stoves but not of passive smoking on children. In the same study, the reverse was shown with different analyses of further data (59), but there were inconsistencies between different cohorts, even in the same city. Dodge (8, 9) examined children's lung function cross-sectionally and logitudinally; he did not find any effect of gas stove usage or passive smoking, although self-reported cough was higher in children exposed to parental smoking. Studies by Binder *et al.* and Schilling *et al.* (2, 49) found indoor levels of RSP higher in Connecticut homes with smokers, but no respiratory effects were seen in adults or children. Often, results have been inconsistent within the same study, or a single positive finding has occurred without consideration for the number of comparisons examined.

The 1983 Geneva Workshop on Environmental Smoke (47) concluded that no definitive studies of acute effects in children have been performed. Furthermore, there was difficulty in interpreting several of the studies due to the large numbers of children involved and the qualitative responses found. Additionally the specific mechanisms of some irritant gases [such as sulfur dioxide ($SO_2$), ozone ($O_3$), and formaldehyde] involved are also unknown.

Retrospective studies of the etiology of respiratory diseases, such as asthma (13), have been discounted because of the major problem of reporting bias (4, 21). Patients with allergies appear to complain more of negative responses to environmental tobacco smoke (35, 40, 45, 54), as do many nonsmokers, but subjective responses generally have not been validated by epidemiological studies. The question also has been raised as to whether levels of environmental smoke per se found in the workplace could be considered sufficient to produce lung function effects in workers (58). There has been little evidence that passive smoking has an effect on chronic symptoms in adults (33, 47).

In Tucson, we have attempted to investigate the relationship between peak flow and reported respiratory symptoms and passive smoking, air pollutants, and aeroallergens (pollen, bacilli, fungi, algae) in the microenvironment of the home as well as the macro- (ambient) environment.

## METHODS

A total of 117 families (229 adult and child subjects) in Tucson were randomly

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chosen in a stratified sample of families in uniform geographic clusters from a representative community population [already under study (20)], as previously described (23, 26). Studies of the total population sample indicated the presence of relationships between weekly symptoms and both air pollutants and allergens, controlling for weather (44). Stratification ensured that the families were representative of families with and without asthma in their geographic clusters. They were studied over a 2-year period, using daily symptom diaries and peak flow meters; daily response rates and peak flows were acceptable for a majority of days in all seasons (23, 28). All families provided environmental information as to characteristics of the house, yard, and streets, heating, cooling, type of stove, water heater, washer and dryer, and smoking in the home (26).

Daily ambient monitoring data for air pollutants, aero-allergens, and meteorological variables with regional and geographic clusters were used (23). Indoor and yard (micro-outdoor) monitorings were conducted in a random cluster sample representative of all study households (23, 26) for particulate matter [RSP and total suspended particulate matter (TSP)], pollen, bacilli, fungi, and algae, for 72 hr each, and grab samples were made for CO and $O_3$. Microenvironmental sampling of pollen, algae, and fungi have been described previously (23, 37, 38). Temperature and humidity varied by season, but the former did not vary very much indoors over the year and the latter did not differ much between indoors and outdoors over the year.

TSP indoors ranged from 5.7 to 68.5 $\mu g/m^3$, and simultaneous micro-outdoor TSP ranged from 2.1 to 169.6 $\mu g/m^3$. Cyclone measurements of RSP had a maximum of 49.8 $\mu g/m^3$ indoors and 124.5 $\mu g/m^3$ outdoors. Electron microscopy yielded little identifying information on the type of dust found on the floors of homes. Infiltration of outdoor TSP indoors was similar to pollen infiltration: total micro-indoor pollen counts were approximately 5% of the micro-outdoor pollen counts, which were about 5% of macro-pollen rates. Both TSP and RSP were significantly associated with environmental tobacco smoke (Table 1), but not with gas stove usage (26).

Averages of the two spot CO readings using infrared determinations were lower than 2.4 ppm; micro-outdoor readings were less than 3.8 ppm. Indoor CO was significantly associated with gas stove usage (26), but not with environmental tobacco smoke (Table 1), nor with gas-burning furnaces, water heaters, or washer–dryers (that is, vented appliances). Although low, it was used as the indicator pollutant measure of combustion emission from the gas stoves. Neither environmental tobacco smoke nor gas stove usage was associated with social status in this sample.

## RESULTS

We found many interactions between indoor and indoor–outdoor pollutants, and weather, with respect to health effects. Children's standardized peak flow (PEF) was related to outdoor environmental factors only ($O_3$, TSP, temperature, and relative humidity); outdoor factors affected nonsmokers more than passive smokers (Table 2).

Adult PEF was related to active smoking (as were chronic symptoms), gas

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TABLE 1
TOTAL SUSPENDED PARTICULATES (TSP), RESPIRABLE SUSPENDED PARTICULATES (RSP), AND CARBON MONOXIDE (CO) BY ENVIRONMENTAL TOBACCO SMOKE

| | ETS | |
|---|---|---|
| | No | Yes |
| TSP ($\mu g/m^3$) | | |
| Mean | 17.7 | 33.0* |
| SD | 10.7 | 18.2 |
| *N* | 14 | 19 |
| RSP ($\mu g/m^3$) | | |
| Mean | 7.2 | 13.1** |
| SD | 8.0 | 13.1 |
| *N* | 15 | 19 |
| CO ($\mu g/m^3$) | | |
| Mean | 1.2 | 1.2 |
| SD | 0.6 | 0.6 |
| *N* | 14 | 12 |

* Difference by two-tailed *t* test, $P < 0.005$.
** Borderline significant (two-tailed *t* test) $0.05 < P < 0.10$.

stove use, and outdoor $NO_2$ (independent and interactive) in asthmatics, allergics, and normals. In some cases, PEF was related to outdoor $O_3$, temperature, and relative humidity in nonnormal groups combined.

PEF in adult asthmatics was related also to temperature, high relative humidity, micro-pollen, and indoor TSP (Table 3), but not to passive smoking. "Asthma attacks" in adult asthmatics were related to temperature and to indoor TSP and RSP, but not to passive smoking or outdoor TSP (28).

Indoor TSP (but not passive smoking) was associated with rhinitis in asthmatics and nonasthmatics, as was RSP. Eye irritation in all adults and increased productive cough in nonasthmatics also were related to indoor TSP, but not to passive smoking. In addition, gas stove usage (and indoor CO) was associated with increased rhinitis in all adults, increased productive cough in asthmatics and allergics, increased sore throat in nonasthmatics, and eye irritation in all adults. Micro-pollen was associated also with adult eye irritation and rhinitis (in summer).

TABLE 2
CHILDREN'S MEAN PEAK FLOWS (EXPRESSED AS STANDARDIZED DEVIATION SCORES[a]: EFFECT OF OUTSIDE OZONE IN PASSIVE AND NONSMOKERS (PERSON DAYS = 1,566)

| | Outside ozone | | | |
|---|---|---|---|---|
| | Very low | Low | Moderate | High[b] |
| Nonsmokers[c] | 0.075 | 0.059 | −0.175 | −0.484[d] |
| Passive smokers | 0.071 | −0.041 | −0.046 | −0.079 |

*Note.* Overall ANOVA $P < 0.0001$.
[a] Deviations of individual values from seasonal means, standardized for seasonal variation.
[b] High: Max hourly $O_3$ (ppm) > 0.08.
[c] Analysis of variance, $P < 0.001$.
[d] $\Delta(\%) = -18 \pm \%$, significantly greater day-to-day change than seen in normals (22).

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TABLE 3
ASSOCIATIONS OF INDOOR TSP WITH AVERAGE PEAK FLOWS (STANDARDIZED DEVIATION SCORES) IN ADULT ASTHMATICS

| PEF | Indoor TSP | | |
|---|---|---|---|
| | Low | Moderate[a] | All |
| Mean | −1.17 | −1.92 | −1.56 |
| SD | 0.99 | 1.83 | 1.54 |
| Person days | 1,205 | 1,334 | 2,539 |

*Note.* Analysis of variance, $P < 0.0001$. Deviations of individual values from seasonal means, standardized for seasonal variation.
[a] 25+ μg/m³.

Thus, sensitive groups (children, asthmatics, allergics, AOD subjects) had more reactions to indoor and outdoor pollutants. Children were found to spend more time outdoors, usually with exercise, and were affected by outdoor pollutants more; these results confirm previous findings in Tucson (19). Indoor pollutants affected sensitive adults more than did outdoor pollutants. Indoor TSP was associated with health effects, but not passive smoking per se, indicating that other characteristics of indoor TSP were important (28).

We have shown that active smokers have higher circulating immunoglobulin E (IgE), which with smoking is related to airway obstructive diseases (3). However, we were not able to see any increase in IgE in passive-smoking children (Table 4). Generally, our recent findings are compatible with our previous negative findings (21, 22, 33). However, the blunting of irritant effects in children (seen in Table 2) needs further explanation.

## DISCUSSION

Other effects of passive smoking have been reviewed recently (27, 47). The studies performed so far continue to provide confusing results vis-à-vis most public health issues. Many of the problems of positive studies are due to poor study design related to reporting bias, lack of control for family concordance, interaction among various pollutant emitters, lack of appropriate measurements (61), and crude statistical studies of geographical differences.

Confounding factors need to be incorporated more explicitly and completely.

TABLE 4
CIRCULATING LOG IMMUNOGLOBULIN E (IgE) IN CHILDREN BY CURRENT PARENTAL SMOKING

| Parental smoking | N | Log IgE | |
|---|---|---|---|
| | | Mean | SD |
| Both smoke | 58 | 1.76 | 0.77 |
| Father smokes | 86 | 1.85 | 0.69 |
| Mother smokes | 28 | 2.09 | 0.64 |
| Neither smoke | 184 | 1.91 | 0.80 |

*Note.* Analysis of variance, $P = 0.258$.

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Better ascertainment and control of the covariables and confounders are needed (12). These include the following:

(a) Unvented combustion products from combustion appliances: gas (especially stoves), wood burning, and kerosene burning.

(b) Effects of other indoor pollutants (e.g., formaldehyde, consumer products), mostly irritant in nature; including interaction effects.

(c) Other indoor conditions of biological importance, such as (i) temperature and humidity conditions, size of house, ventilation (e.g., air exchange rates); (ii) pollen, molds, mites, and other allergens; and (iii) infectious organisms.

(d) Outdoor and occupational exposures and personal mobility, representing total/integrated exposure (includes consideration of geographic region, urban/rural setting).

(e) Socioeconomic status, cultural, ethnic, and religious factors (re: crowding, number of siblings, household conditions, occupation of parents, host conditions, risk of infectious spread, medical knowledge, medical care, reporting biases, etc.).

(f) Demographic and medical characteristics of the study population, such as (i) age, sex, marital status; (ii) presence of underlying respiratory condition (e.g., bronchial reactivity, clinical airways obstruction, atopy); (iii) disability and/or comorbidity.

(g) Parental syndromes, like productive cough, which would mask other processes/mechanisms (e.g., genetic, infectious) or affect reporting (4, 21) (e.g., winter morning phlegm).

(h) Maternal smoking during pregnancy and resulting effects on fetus and newborn, including low birth weight (33, 46).

(i) Annoyance responses, including odor problems, other psychological or social responses to tobacco smoking in a nonsmoker.

(j) Unknown smoking by subject.

A major problem to be resolved is the determination of the actual dose to the passive smoker (34, 61, 62). Monitoring of airborne particulates and organics is needed (47). Respirable particulates indoors are usually driven by smoking (2, 26, 64). However, the smoking-generated fraction of these concentrations is not related to health effects (28, 49). This implies that sizing and speciation of particles are necessary (61, 62). The differentiation of sources and concentrations would be very useful in determining mechanism of effects as well as the sources of primary concern.

Friedman and colleagues (12) showed, by questionnaire responses of about 38,000 adults, that 63% of people were exposed to environmental tobacco smoke, but their reported exposure was poorly correlated with serum thiocyanate levels. Exposures were correlated significantly with other health-related characteristics, including age, use of alcohol and marijuana, and exposure to occupational hazards. They were also correlated with sex, race, lower educational status, and single marital status. The reported concordance of marital smoking habits by categorization of spouse's smoking resulted in considerable misclassification, indicating spouse's smoking habits are a very inaccurate index of exposure. Future studies of passive-smoking effects should thus take these findings into account.

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The WHO/EURO Working Group (62) examined passive smoking within the context of all indoor exposures. It was felt that the current levels of knowledge did not yet allow quantitative assessment of the impact on the public health, as the knowledge of exposure effect relationships was inadequate, especially for delayed effects of chronic exposures. In terms of exposure levels, there were no data on how many cigarettes were being smoked in each of those indoor places where passive smoking can occur, and the ability to estimate how this is distributed within the building stock is marginal. It was felt that the amount of current information about the fraction of the population exposed to levels high enough to be a public health concern is smaller. The amount of information about the specific pollutants that might be responsible for the effects of concern is also very small. Of the variety of potential adverse effects, it was felt that only for irritation, odor, and airway response is there even a marginal amount of current knowledge. This group also felt that there were many possible pollutant interactions and that there were many other confounding exposures involved in examining the effects of passive smoking. Thus, in terms of relative priority, effects of passive smoking were low in comparison with other concerns, based on knowledge levels. Since, however, passive smoking may indeed be an important public health concern, it should be studied further. Attempts are currently being made by various groups to consider potential efficacious methods for further studies of the health effects of passive smoking (33, 47).

## REFERENCES


1. Anderson, H. R. Respiratory abnormalities, smoking habits, and ventilatory capacity in a highland community in Papua, New Guinea: Prevalence and effect on mortality. *Int. J. Epidemiol.* 8, 127–135 (1979).
2. Binder, R. E., Mitchell, C. A., Husein, H. R., and Bouhuys, A. Importance of the indoor environment in air pollution exposure. *Arch. Environ. Health* 31, 277–279 (1976).
3. Burrows, B., Lebowitz, M. D., Barbee, R. A., Knudson, R. J., and Halonen, M. Interactions of smoking and immunologic factors in relation to airways obstruction. *Chest* 84, 657–661 (1983).
4. Cederlöf, R., and Colley, J. Epidemiological investigations on environmental tobacco smoke. *Scand. J. Respir. Dis. Suppl.* 91, 47–49 (1974).
5. Chapman, R. S., Hasselblad, V., Hayes, C. G., Williams, J. V. R., and Hammer, D. I. Air pollution and childhood ventilatory function. I. Exposure to particulate matter in two southeastern cities, 1971–1972, *in* "Clinical Implications of Air Pollution Research" (A. J. Finkel and W. C. Duel, Eds.), pp. 285–303. Publ. Sci. Group, Inc., Acton, Mass., 1976.
6. Comstock, G., Meyer, M. B., Helsing, K. J., and Tockman, M. S. Respiratory effects of household exposures to tobacco smoke and gas cooking. *Amer. Rev. Respir. Dis.* 124, 143–148 (1981).
7. Dahms, T. E., Bolin, M. D., and Slavin, R. G. Passive smoking: Effects of bronchial asthma. *Chest* 80, 530–534 (1981).
8. Dodge, R. R. The effects of indoor air pollution on Arizona children. *Arch. Environ. Health* 37, 151–155 (1982).
9. Dodge, R. R. The respiratory health and lung function of Anglo-American children in a smelter town. *Amer. Rev. Respir. Dis.* 127, 158–161 (1983).
10. Ekwo, E. E., Weinberger, M. M., Lachenbruch, P. A., and Huntley, W. H. Relationship of parental smoking and gas cooking to respiratory disease in children. *Chest* 84, 662–668 (1983).
11. Florey, C. du Ve, Melia, R. J. W., Chinn, S., Goldstein, B. D., Brooks, A. G. F., John, H. H., Brughead, L. B., and Webster, X. The relation between respiratory illness in primary school


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children and the use of gas for cooking. III. Nitrogen dioxide, respiratory illness and lung function. *Int. J. Epidemiol.* 8, 347–353 (1979).

12. Freelman, G. D., Petitti, D. B., and Buwol, R. D. Prevalence and correlates of passive smoking. *Amer. J. Public Health* 73, 401–405 (1983).
13. Gortmaker, S. L., Walker, D. K., Jacobs, F. H., and Ruch-Ross, H. Parental smoking and the risk of childhood asthma. *Amer. J. Public Health* 72, 574–579 (1982).
14. Hammer, D. I., Miller, F. J., Stead, A. G., and Hayes, C. G. Air pollution and childhood ventilatory function. I. Exposure to particulates matter in two southeastern cities, 1971–1972. *in* "Clinical Implications of Air Pollution Research" (A. J. Finkel and W. C. Duel, Eds), pp. 321–337. Publishing Sciences Group, Acton, Mass., 1976.
15. Hasselblad, V., Humble, C. G., Graham, M. G., and Anderson, H. S. Indoor environmental determinants of lung function in children. *Amer. Rev. Respir. Dis.* 123, 479–485 (1981).
16. Hiller, F. C., McCusker, K. T., Mazumber, M. K., Wilson, J. D., and Bone, R. C. Deposition of sidestream cigarette smoke in the human respiratory tract. *Amer. Rev. Respir. Dis.* 125, 406–408 (1982).
17. Kauffmann, F., and Brille, D. Respiratory function in passive smokers' children, *in PROC IEA*, Edinburgh, August 1981.
18. Kauffmann, F., Tessier, J. F., and Oriol, P. Adult passive smoking in the home environment: A risk factor for chronic airflow limitation. *Amer. J. Epidemiol.* 117, 269–280 (1983).
19. Lebowitz, M. D., Baadheim, P., Cristea, G., Martowitz, D., Miniuszek, J., Stuniec, M., and Van Wyck, D. The effect of air pollution and weather on lung function in exercising children and adolescents. *Amer. Rev. Respir. Dis.* 109, 262–273 (1974).
20. Lebowitz, M. D., Knudson, R. J., and Burrows, B. The Tucson epidemiology study of chronic obstructive lung disease. I. Methodology and prevalence of disease. *Amer. J. Epidemiol.* 102, 137–152 (1975).
21. Lebowitz, M. D., and Burrows, B. Respiratory symptoms related to smoking habits of family adults. *Chest* 69, 48–50 (1976).
22. Lebowitz, M. D., Armet, D. B., and Knudson, R. The effect of passive smoking on pulmonary function in children. *Environ. Int.* 8, 371–374 (1982).
23. Lebowitz, M. D., O'Rourke, M. K., Dodge, R., Holberg, C. J., Corman, G., Hoshaw, R. W., Pinnas, J. L., Barbee, R. A., and Sneller, M. R. The adverse health effects of biological aerosols, other aerosols, and indoor microclimate on asthmatics and non-asthmatics. *Environ. Int.* 8, 375–380 (1982).
24. Lebowitz, M. D. *Cong. Rec.*, 97th Congress, 2nd session, 16 December 1982, E5253.
25. Lebowitz, M. D., Knudson, R. J., Robertson, G., and Burrows, B. Significance of intraindividual changes in maximum expiratory flow volume and peak expiratory flow measurements. *Chest* 81, 566–570 (1982).
26. Lebowitz, M. D., Holberg, C. J., O'Rourke, M. K., Corman, G., Dodge, R. R. Gas stove usage, CO and TSP, and respiratory effects, *in* "Proceedings: Indoor Air Pollutants—Health Effects." Air Pollu. Control Association, Pittsburgh, 1983.
27. Lebowitz, M. D., Health effects of indoor pollution. *Annul. Rev. Public Health* 4, 203–221 (1983).
28. Lebowitz, M. D. The effects of environmental tobacco smoke exposure and gas stoves on daily peak flow rates in asthmatic and non-asthmatic families. *Eur. J. Respir. Dis.* 65 (Suppl. 133), 190–195 (1984).
29. Martinez, F., and Ronchetti, R., *in* "Proceedings of the National Congress on Pediatric Bronchopneumonology" (D. Castello, Ed.). Italian Ped. Bronchopneumonol. Association, Turin, 1982.
30. Melia, R. J. W., Florey, C. du Ve, Altman, D. G., and Swan, A. V. Association between gas cooking and respiratory disease in children. *Brit. Med. J.* 2, 149–152 (1977).
31. Melia, R. J. W., Florey, C. du Ve, Morris, R. W., Goldstein, B. D., Clark, D., and John, H. H. Childhood respiratory illness and the home environment. I. Relations between nitrogen dioxide, temperature, and relative humidity. *Int. J. Epidemiol.* 11, 155–163 (1982).
32. Melia, R. J. W., Florey, C. du V., Ahman, D. G., and Swan, A. V. The association between respiratory illness and $NO_2$, temperature and relative humidity. *Int. J. Epidemiol.* 11, 164–169 (1982).

2023383714

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33. National Heart Lung and Blood Inst. "Report of a Workshop on the Respiratory Effects of Involuntary Smoking: Epidemiological Studies." U.S. Govt. Printing Office, Washington, D.C., December 1983.
34. National Research Council (NRC). "Indoor Pollutants." National Academy of Sciences, Washington, D.C., 1981.
35. O'Connell, E. J., and Logan, G. B. Parental smoking in childhood asthma. *Ann. Allergy* 32, 142–145 (1974).
36. Olansky, S. J. Is smoker/nonsmoker segregation effective in reducing passive inhalation among nonsmokers? *Amer. J. Public Health* 72, 737–739 (1982).
37. O'Rourke, M. K. Pollen dispersal and its relationship to respiratory illness, *in* "Proceedings of the First International Conference on Aerobiology" (German), pp. 81–88. Sympusium Publishers, Munich, 1980.
38. O'Rourke, M. K., and Lebowitz, M. D. A comparison of regional atmosphere pollen with pollen collected at and near homes. *Grana* 22, 1–10 (1984).
39. Pimm, P. E., Silverman, F., and Shephard, R. J. Physiological effects of acute passive exposure to cigarette smoke. *Arch. Environ. Health* 33, 201–213 (1978).
40. Pipes, D. M. Allergy to tobacco smoke. *Ann. Allergy* 28, 277–82 (1945).
41. Poluk, E. Le Papier a cigarette. Son Role Dans la Pollution de Lieux Habites. Tabagisme Passif: Notion Nouvelle Precisee. *Bruxelles-Med.* 57, 335–340 (1977).
42. Radford, E. P., and Drizd, T. A. Blood carbon monoxide levels in persons 3–74 years of age: United States, 1976–80. *NCHS Advance Data* 76, 1–24 (1982).
43. Repace, J. L., and Lowrey, A. H. Indoor air pollution tobacco smoke, and public health. *Science (Washington, D.C.)* 208, 464–472 (1980).
44. Robertson, G., and Lebowitz, M. D. Analysis of relationshhips between symptoms and environmental factors over time. *Environ. Res.* 33, 130–143 (1984).
45. Romer, J., and Hermann, H. Tobaksrygnings Betydning for asthma og Rhinitis. *Ugeskr. Laeg.* 145, 1025–1027 (1983).
46. Rylander, R. Environmental tobacco smoke effects on the nonsmoker: Report from a workshop. University of Geneva, 1974. *Scand. J. Respir. Dis.* (Suppl. 91). (1974).
47. Rylander, R. (Ed.). Environmental tobacco smoke: effects and exposure levels: Proceedings of a workshop. *Eur. J. Respir. Dis.* 65 (Suppl. 133), 1–52 (1984).
48. Schenker, M. B., Samet, J. M., and Speizer, F. E. Risk factors for childhood respiratory disease: The effects of host factors and home environmental exposures. *Amer. Rev. Respir. Dis.* 128, 1038–1043 (1983).
49. Schilling, R. S. F., Letai, A. D., Hui, S. L., Beck, G. J., Schoenbert, J. B., and Bouhuys, A. Lung function, respiratory disease, and smoking in families. *Amer. J. Epidemiol.* 106, 274–283 (1977).
50. Shephard, R. J., Collins, R., and Silverman, F. Passive exposure of asthmatic subjects to cigarette smoke. *Environ. Res.* 20, 392–402 (1972).
51. Shephard, R. J., Collings, R., and Silverman, F. Responses of exercising subjects to acute "passive" cigarette smoke exposure. *Environ. Res.* 19, 279–291 (1979).
52. Simecek, C. Reflection of passive exposure to smoking in the home on the prevalence of chronic bronchitis non-smokers. *Czech. Med.* 3, 308–310 (1980).
53. Sofoluwe, G. O. Smoke pollution in dwellings of infants with broncho-pneumonia. *Arch. Environ. Health* 16, 670–672 (1968).
54. Speer, F. Tobacco and the non-smoker: A study of subjective symptoms. *Arch. Environ. Health* 16, 443–446 (1968).
55. Speizer, F. E., Ferris, B., Jr., Bishop, Y. M. M., and Spengler, J. Health effects of indoor $NO_2$ exposures: Preliminary results, *in* "Nitrogen Oxides and Their Effects on Health" (S. F. Lee, Ed.), pp. 343–359. Ann Arbor Press, Ann Arbor, Mich., 1980.
56. Speizer, F. E., Ferris, B., Jr., Bishop, Y. M. M., and Spengler, J. Respiratory disease rates and pulmonary function in children associated with $NO_2$ exposure. *Amer. Rev. Respir. Dis.* 121, 3–10 (1980).
57. Tager, I. B., Weiss, S. T., Rosner, B., and Speizer, F. E. Effect of parental cigarette smoking on the pulmonary function of children. *Amer. J. Epidemiol.* 110, 15–25 (1979).

2023383715

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58. Valentin, H., Bost, H.-P. and Wawra, E. Das Passivrauchen am Arbeitsplatz-eine Gesundheitsschädigung? Zentralblatt fur Bakteriologie, Parasitenkunde, Infektionskrankheiten und Hygiene; Erste Abteilung: Originale, Reihe B: Hygiene. *Präventive Medizin* 167, 405–434 (1978).
59. Ware, J. H., Dockery, D. W., Spiro, A., III, Speizer, F. E., and Ferris, B. G., Jr. Passive smoking, gas cooking, and respiratory health of children living in six cities. *Amer. Rev. Respir. Dis.* 129, 366–374 (1984).
60. White, J., and Froeb, H. Small-airways dysfunction in non-smokers chronically exposed to tobacco smoke. *New Engl. J. Med.* 302, 720–723 (1980).
61. World Health Organization (WHO). 1982. "Estimating Human Exposure to Air Pollutants." WHO, Copenhagen/Geneva, 1982.
62. WHO/EURO. "Indoor Air Pollutants: Exposure and Health Effects Assessments." EURO Publ. No 78. World Health Organization, Copenhagen, 1983.
63. Yarnell, J. W. G., and St. Leger, A. S. Respiratory illness, maternal smoking habit and lung function in children. *Brit. J. Dis. Chest* 73, 230–236 (1979).
64. Yocom, J. E. Indoor–outdoor air quality relationships: A critical review. *J. Air Pollut. Control Assoc.* 32, 500–520 (1982).

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PREVENTIVE MEDICINE 13, 656–669 (1984)

# The Influence of Passive Smoking on Pulmonary Function—A Study of 1,351 Office Workers[1]

MICHAEL KENTNER,[2] GERHARD TRIEBIG, AND DIETER WELTLE

*Institute of Occupational and Social Medicine and Polyclinic of Occupational Diseases of the University of Erlangen–Nuremberg, Schillerstrasse 25/29, D-8520 Erlangen, Federal Republic of Germany*

Until now it has been difficult to ascertain how much passive inhalation of tobacco smoke affects bronchopulmonary function. To answer this question, an investigation involving 1,351 white collar workers was carried out. Information about active and passive tobacco smoke exposure was obtained by a standardized questionnaire. This made it possible to subdivide the overall group into five subgroups: Never smokers, passive smokers, ex-smokers, current smokers, and other smokers. Forced expiratory vital capacity (FVC) and maximal expiratory flow-volume (MEFV) curves were used for lung function analysis. From these curves FVC, forced mid-expiratory flow (FEF 25/75), forced end-expiratory flow (FEF 75/85), and maximal mid-expiratory flow (MEF 25/75) were determined and standardized for sex, age, height, and body weight. Passive smokers evaluated by this method showed essentially no decrease in parameters describing ventilatory function. It is concluded from the dose- and time-effect relationships obtained in active smokers between the lung function parameters and the duration of tobacco smoke exposure on the one hand and the daily consumption of cigarettes on the other that passive smoking in small doses may have no essential effect on pulmonary function. © 1984 Academic Press, Inc.

## INTRODUCTION

One of the numerous questions that have not yet been unequivocally answered with respect to passive smoking is its effect on bronchopulmonary function in people with healthy lungs. A number of studies in children indicate a certain negative influence, particularly in children of preschool age (11, 30, 33). Other studies, however, fail to confirm such an association (5, 18, 27, 29). Recent reviews on this subject can be found in publications by Weiss *et al.* (34) and by Lebowitz (19).

To date, only a few studies have been published on the effects of passive smoking and pulmonary function in adults (7, 14, 27, 35). These investigations were usually initiated within the framework of research projects not primarily concerned with passive smoking. As a result, certain restrictions must inevitably be made regarding the relevance of these findings. This point will be considered in more detail in the discussion. The investigation reported herein, in contrast, was designed and carried out for the sole purpose of confirming or denying any significant effects of passive smoking on the bronchopulmonary system using pulmonary function measurement.

[1] Presented at the Symposium "Medical Perspectives on Passive Smoking," April 9–12, 1984, Vienna, Austria.

[2] To whom reprint requests should be addressed.



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## PATIENTS AND METHODS

A total of 1,351 people (941 men and 410 women) took part in the study, which was carried out in 1982–1983. Participation in the study was voluntary. All participants were white-collar workers, the majority of whom were employed in sedentary office jobs. Test subjects were recruited from the staff of an administration authority and two large industrial companies located in Northern Bavaria, a region with a relatively low level of air pollution. All those participating anonymously filled out a standardized questionnaire specially developed for this study, and upon completion handed it in a closed envelope to the person carrying out the examinations.

The pulmonary function tests were carried out with an electronic spirometer.[3] The device was calibrated either daily or prior to each new series of measurements. The spirometric examination was conducted by two trained examiners with a knowledge of medicine. Special attention was paid to good cooperation on the part of the test subject with respect to the respiration exercises, which were generally repeated two to three times.

Of the various pulmonary function parameters, those listed in Table 1 were included in the further analysis. These parameters were identical to those used in comparable studies.

These data were transferred, anonymously, to documents designed for electronic data processing. Evaluation of the data was effected on a data processing system of the type Cyber 845.[4] After various plausibility checks and appropriate adjustment of the data record and group, the overall group was subdivided into five subgroups (Fig. 1):

(a) *Never smokers* (*NS*), defined as persons who have never been regularly exposed to tobacco smoke, either actively or passively.

(b) *Passive smokers* (*PS*), defined as subjects who have never actively smoked, but who are currently exposed to passive smoking. Here, three subgroups were differentiated:

(i) PS exclusively with household exposure to passive smoking (PS/H),
(ii) PS exposed passively to tobacco smoke only at their place of work (PS/W), and
(iii) persons with a combination of passive smoke exposure (PS/HW).

(c) *Ex-smokers* (*ES*), defined as persons who had given up active tobacco smoking at least 6 months previously.

(d) *Current smokers* (*CS*), representing the group of persons who, at the time of the investigation were actively smoking and inhaling the smoke.

(e) *Other smokers* (*OS*), representing a remaining category in which noninhaling cigarette smokers and cigar and pipe smokers were grouped together.

Considerable care was expended on the standardization of the analytical pulmonary function data. For all the pulmonary function parameters, on the basis of the NS group, formulae for internal predicted values were computed, using

[3] Siregnost FD10, XY Recorder E2218, Siemens, Erlangen, FRG.
[4] Control Data Corporation, Minneapolis, Minn.

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TABLE 1
THE PARAMETERS EMPLOYED FOR PULMONARY FUNCTION ANALYSIS

| Method | Pulmonary function parameter | Abbreviation |
|---|---|---|
| Forced vital capacity curve | Forced expiratory vital capacity | FVC |
| | Forced mid-expiratory flow | FEF 25/75 |
| | Forced end-expiratory flow | FEF 75/85 |
| Maximal expiratory flow–volume curve | Maximal mid-expiratory flow | MEF 25/75 |

multiple regression analyses, and taking into account sex, age, height, and body weight. A comparison of the reference values thus obtained, with standard values established by other authors (4, 17, 23, 26) shows a good level of agreement. According to these figures, the standard values decrease with increasing age and weight, while an increase in height results in an elevation in the predicted value (Table 2). All the individual values actually measured were converted into a percentage deviation from the predicted standard value.

The differences in the pulmonary function parameters standardized in this way were statistically checked in a subgroup comparison, using a nonparametric test procedure. This procedure involved a comparison of several independent samples as described by Kruskal and Wallis (36). A significance level of $P \leq 0.05$ was chosen.

## RESULTS

### *Group Structure*

Table 3 shows the size, average age, and prevalence of pulmonary disease in

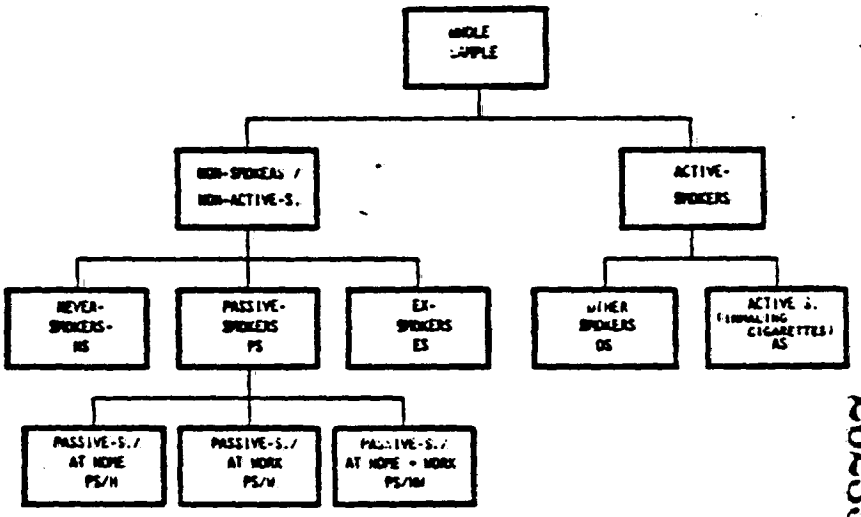


FIG. 1. Subgroups in accordance with tobacco smoke exposure criteria.

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TABLE 2
CORRECTION FACTORS FOR THE STANDARDIZATION OF THE PULMONARY FUNCTION PARAMETERS

| | | Age (years) | Height (m) | Weight (kg) | Const. |
|---|---|---|---|---|---|
| FVC | M | −0.028 | 5.71 | −0.011 | −3.37 |
| (L) | F | −0.038 | 4.87 | −0.003 | −2.94 |
| FEF 25/75 | M | −0.020 | 4.30 | −0.014 | −1.72 |
| (L/S) | F | −0.043 | 1.37 | −0.020 | 1.27 |
| FEF 75/85 | M | −0.027 | 13.85 | −0.048 | −12.01 |
| (L/S) | F | −0.091 | 6.48 | −0.018 | −0.36 |
| MEF 25/75 | M | −0.006 | 3.51 | −0.029 | −0.03 |
| (L/S) | F | −0.023 | 1.84 | 0.031 | 0.03 |

each subgroup. The average age varies between 39 and 43 years for the men, and between 29 and 41 years for the women. Since almost all the pulmonary function parameters correlated negatively with age, the need for a standardization was thus underscored.

The CS group was most frequently affected by bronchopulmonary prior diseases, but the differences were not significant when compared with the other groups. Since on the one hand, passive inhalation of tobacco smoke may induce, or at least aggravate, chronic bronchitis, while active smoking may be the cause of this disease, the analysis of pulmonary function parameters would be affected by both including or excluding bronchitic subjects. It was, however, found that this had essentially no influence on the median values or the regression analysis. For this reason, a basic group, excluding subjects with bronchopulmonary diseases such as pneumonia, asthma, and tuberculosis in their history, but including the bronchitics, was used for further evaluation.

TABLE 3
STRUCTURE OF THE GROUP INTO SUBGROUPS. NUMBER OF SUBJECTS, AVERAGE AGE, AND BRONCHOPULMONARY PRIOR DISEASES

| | No. of subjects examined | | Age (median values) | | Pneumonia, bronchial asthma, tuberc. of the lungs | | Chronic bronchitis | |
|---|---|---|---|---|---|---|---|---|
| | M | F | M | F | M | F | M | F |
| NS | 142 | 66 | 42 | 41 | 21 | 7 | 3 | 2 |
| PS | 251 | 132 | 40 | 36 | 30 | 17 | 8 | 3 |
| ES | 301 | 99 | 43 | 39 | 42 | 8 | 9 | 3 |
| OS | 64 | 12 | 41 | 29 | 5 | 2 | 1 | 2 |
| CS | 183 | 141 | 39 | 31 | 28 | 11 | 9 | 6 |
| Su. | 941 | 410 | 41 | 35 | 136 | 45 | 30 | 18 |

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*Pulmonary Function Parameters in a Comparison of the Subgroups*

In the case of FVC (Fig. 2), the median values for men are similar in all five subgroups, while, in women, only that of the CS group differs significantly from the three groups of nonsmokers.

The comparison of FEF 25/75 (Fig. 3) values shows results similar to those seen for FVC, as do the results of $FEV_1$, not shown here. The greatest negative deviation of the medians from the predicted FEF 25/75 values in both sexes was found in the CS group. However, no statistically significant differences were seen between the groups, which revealed a considerable scatter range for this parameter.

In Fig. 4, the FEF 75/85 values are shown. Here, ES and CS differ significantly both from NS and from PS. This, however, applies only to men.

The MEF 25/75 values show an even greater range of scatter than the prior parameters (Fig. 5). Nevertheless, here, statistically significant differentiation between the individual subgroups is possible. Thus, in the case of the men, the ES group differs from both the PS and CS, while in the women, NS and ES differ from CS.

If we compare the deviations in the predicted values of the pulmonary function parameters among the passive smoker subgroups, a heterogeneous picture is obtained. Here, FEF 25/75 (Fig. 6) and FEF 75/85 (Fig. 7) values are considered by way of example.

For the FEF 25/75 values, all the male PS groups are at about the same level (in the 100% range). By contrast, in the female PS group, a gradation is seen, with PS/W showing the smallest negative deviation from the predicted value, followed by PS/H and PS/HW (Fig. 6).

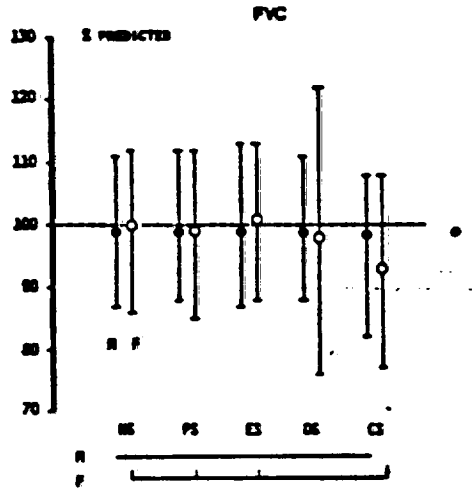


FIG. 2. Medians and 66 % confidence levels of the percentage deviations from predicted values of FVC in sex-separated subgroup comparison.

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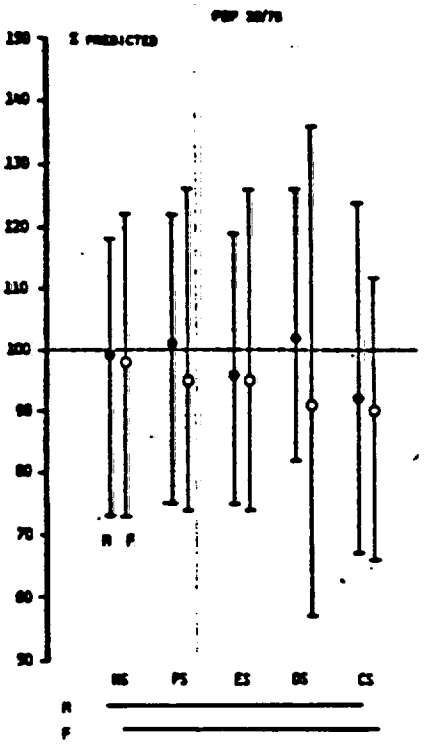


FIG. 3. Medians and 66 % confidence levels of the percentage deviations from predicted values of FEF 25/75 in sex-separated subgroup comparison.

For the FEF 75/85 measurement, the situation is almost the reverse of the FEF 25/75 medians (Fig. 7). Among the men a "ranking" can be observed in that PS/H have the best function figures, followed by PS/W and PS/HW. Among the women, the medians are scattered around the 100% range in all three groups. Neither for FEF 25/75, nor for FEF 75/85, were any statistically significant group-specific differences evident.

*Dose- and Time-Effect Relationships between Tobacco Smoke Exposure and Pulmonary Function*

In the CS group, the individual pulmonary function parameters were correlated with both the duration of tobacco smoke exposure and the daily consumption of cigarettes. The time-effect regressions are briefly discussed for FEF 75/85.

Among men, this parameter deteriorates with increasing duration of tobacco smoke exposure. This relationship is statistically significant at the 5% level (Fig. 8). Among women, however, no statistically significant change in FEF 75/85 as a function of the duration of exposure to tobacco smoke can be seen (Fig. 9).

For parameters FVC, FEF 25/75, and MEF 25/75, dose- and time-effect re-

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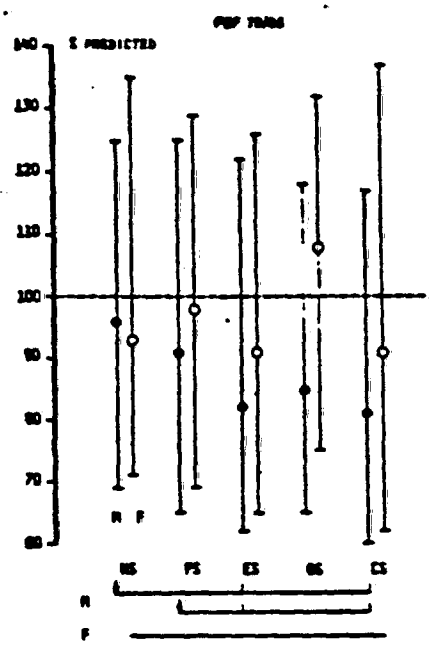


FIG. 4. Medians and 90 % confidence levels of the percentage deviations from predicted values of FEF 75/85 in sex-separated subgroup comparison.

lationships with only low correlation coefficients, not statistically significant, were observed.

## DISCUSSION

Inflammation of the airways is the starting point of all chronic noncarcinogenic bronchopulmonary effects of tobacco smoke inhalation (31). Although the processes which bring about functionally significant changes following the inhalation of tobacco smoke are not yet fully known, it may be assumed that they represent multifactorial processes, for which the following pathomechanisms are under discussion:

(a) Enlargement of the mucous gland mass with resulting hypersecretion (9).

(b) Impairment of the function of the respiratory ciliated epithelium, with build-up of secretion (3).

(c) Increase in bronchial muscles (25), due to reflectory bronchoconstriction or direct bronchospasmic effects (2).

(d) Release of proteolytic enzymes from alveolar macrophages, with consecutive formation of a centro-acinar pulmonary emphysema after years of smoking (9).

(e) "Displacement" of the surfactant factor (25), with possible promotion of atelectasis.

FIG. 5.
MEF 25

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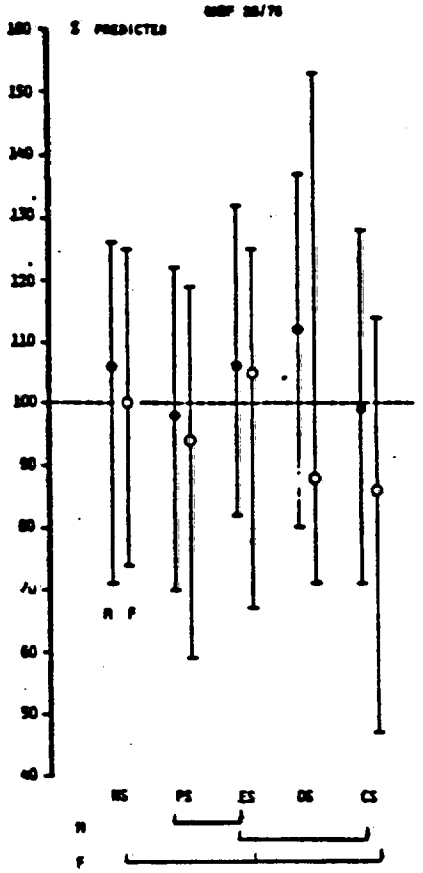


FIG. 5. Medians and 66 % confidence levels of the percentage deviations from predicted values of MEF 25/75 in the sex-separated subgroup comparison.

The inflammation of the small airways represents the initial reaction of the bronchopulmonary system to the active inhalation of tobacco smoke (9, 24). This investigation is primarily concerned with clarifying the question of whether passive exposure to tobacco smoke results in function-analytical significant changes in the small airways. There is a close correlation between structural changes of the small airways and the associated effects on pulmonary function (8), although the prognostic value of these lesions with respect to severe obstructive airway diseases has not yet been established (6, 28).

The function-analytic correlate of a small airways dysfunction is, among other things, the flow limitation in the largely effort-independent, end-expiratory part of vital capacity (20, 21, 28). This results in characteristic deformations of FVC or flow volume curves, which are relatively easy to obtain in an investigation of larger groups of subjects. Although doubts have been expressed (1, 10) on the

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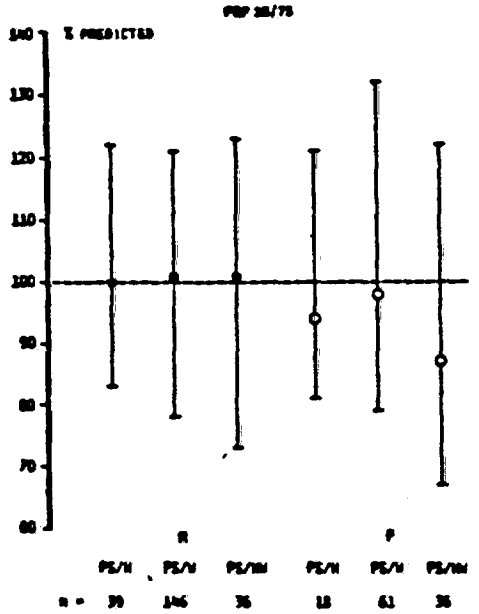


FIG. 6. Medians and 66 % confidence levels of the percentage deviations from predicted values of FEF 25/75 in the sex-separated comparison, for various types of passive smoke exposure.

specificity of such pulmonary function analyses with respect to a small airways dysfunction diagnosis, the view that the determination of parameters such as FEF 25/75, FEF 75/85, and MEF 25/75, in this respect, represents a sensible and useful function-analytic method (6, 22, 28) has largely become accepted. Admittedly, the determination of flow rates or flow differences in the diagnosis of small airways dysfunction makes sense only when normal values for such spirometric parameters as FVC and FEV, are present (10, 22, 28), that is, when significant ventilatory and respiration-mechanical disturbances affecting the large airways can be excluded. This may be assumed, for the present study, since in all subgroups, independent of sex, the FVC values scatter only in the range of a $\pm 2\%$ deviation from the predicted norm. Only the female CS group, at $-7\%$, represents an exception. Similar remarks also apply to the $FEV_1$ parameter.

It is well known that end-expiratory flow determinations are subject to a large intraindividual (6) and interindividual (16) variation spectrum. For this reason, particular care was taken in this study to eliminate as far as possible, such interfering factors as prior bronchopulmonary diseases or inhaled noxae other than tobacco smoke. Furthermore, in order to limit the danger of an evaluation bias, the study was performed under blind conditions and great importance was attached to the precise standardization of the pulmonary function measuring results.

Nevertheless, it was not possible to differentiate all five subgroups defined in accordance with tobacco smoke exposure criteria, on the basis of pulmonary function analysis. It proved possible only to discriminate the CS group from all

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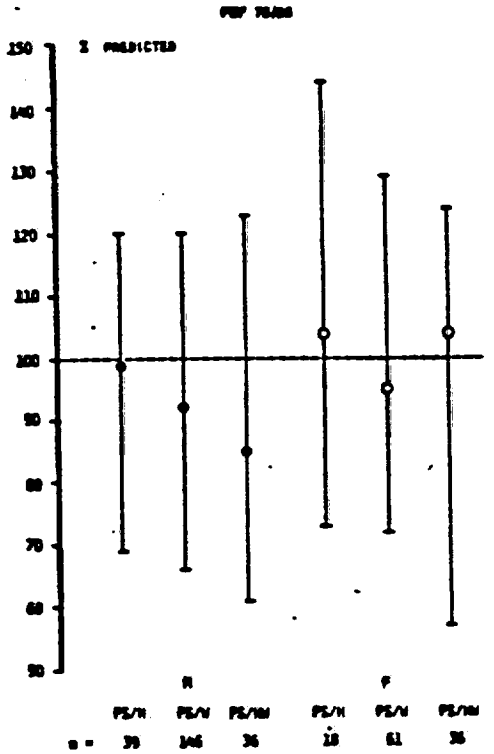


FIG. 7. Medians and 66 % confidence levels of the percentage deviations from predicted values of FEF 75/85 in the sex-separated comparison, for various types of passive smoke exposure.

categories of nonsmokers and from nonactive smokers. In this connection, FEF 75/85 and MEF 25/75 revealed the greatest discrimination. Even time- and dose-effect relationships in the CS group between tobacco smoke exposure and number of cigarettes smoked per day on the one hand, and pulmonary function on the other, revealed only a few statistically significant correlations which, however, revealed inconsistencies in the sex-separated comparison. This result is possibly due to the relatively low average age of the members of the groups investigated, particularly among women.

The precision of the measuring methodology and curve evaluation may be considered good. This was demonstrated by a parallel determination of the flow volume curve for all subjects with the aid of a second electronic spirometer which determined the measured values automatically. It may, therefore, be assumed that the heterogeneity and inconsistency of the pulmonary function pattern results mainly from the large interindividual variability of the measured data. Thus, apparent correlations may also be simulated.

On the basis of the calculations of various authors (12, 13, 15, 32), for passive smokers in general, a daily exposure of the order of magnitude of 0.2 to a max-

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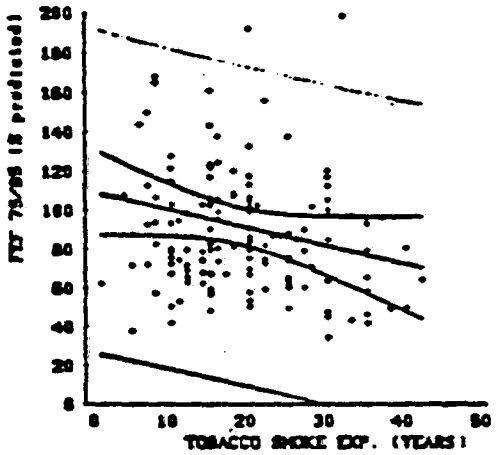


FIG. 8. Regression between FEF 75/85 and tobacco smoke exposure in years in male current smokers ($r = 0.203$, $P = 0.012$).

imum of five cigarette equivalents (depending upon the smoke constituents) may be assumed. In addition, the various types of inhalation on the part of active and passive smokers must be taken into account. While active smokers inhale primarily mainstream smoke constituents via mouth breathing, passive smokers inhale largely sidestream smoke constituents which enter the bronchial system via the nasopharynx. If additional consideration is given to the filtration function of nasal breathing, it may be stated that in the case of passive smoking one is dealing with the effects of a relatively low dose. Since, despite optimized methodology, clear and unequivocal dose- and time-effect relationships between daily cigarette consumption or smoking duration in years and pulmonary function have proved

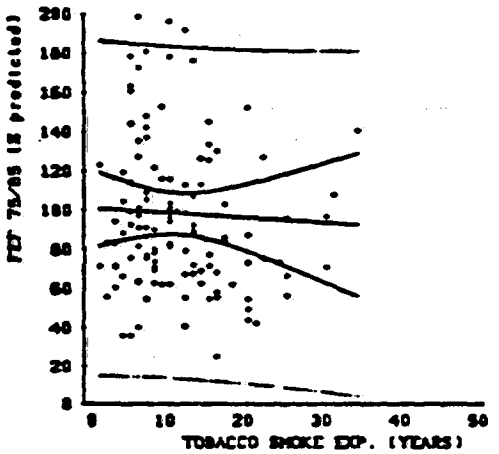


FIG. 9. Regression between FEF 75/85 and tobacco smoke exposure in years in female current smokers ($r = 0.042$, $P = 0.641$).

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impossible to establish or have been established only in an inconsistent form (as in the case of CS), this is all the more likely to be the case for the low-dose situation met with in passive smoking.

As mentioned at the beginning of this article, only a few studies have so far considered the question of possible impairment of pulmonary function in adult passive smokers. These studies all have the disadvantage, owing to the nature of the data collection, of not being able to form groups for all possible factors of active and passive tobacco smoke exposure. This means that there is a danger of selection bias involving the nonconsideration of possible positive or negative results. Since, however, a similar pulmonary function methodology was employed, comparisons between our results and those reported in other studies are possible.

In common with us, Schilling *et al.* (27) and Comstock *et al.* (7) were unable to establish any statistically significant reduction of pulmonary function in passive smokers (Table 4). In contrast, Kauffmann *et al.* (14) found differences between

TABLE 4

REVIEW OF THE LITERATURE ON ARTICLES SO FAR PUBLISHED ON THE TOPIC PULMONARY FUNCTION IN PASSIVE SMOKERS

| Authors | Subgroups* | | Pulmonary function parameter | Results* |
|---|---|---|---|---|
| Schilling *et al.*, 1977 | NS/H<br>PS/H<br>CS | $N$ = 138<br>114<br>78 | FVC, $FEV_1$<br>PEF, MEF 50<br>MEF 25 | No statistically significant reduction of lung function for PS/H. |
| White and Frueh, 1980 | NS<br>PS/W<br>CS | $N$ = 400<br>400<br>1300 | FVC, $FEV_1$<br>FEF 25/75 | Concerning FEF 25/75 and FEF 75/85: PS/W show statistically significantly lower values than NS. Values in PS/W do not differ essentially from values in CS smoking 1–10 cigarettes per day and in CS without inhalation. |
| Comstock | PS/H<br>ES/H<br>CS | $N$ = 539<br>232<br>953 | $FEV_1$<br>$FEV_1$/FVC | No statistically significant reduction of lung function for PS/H. |
| Kauffmann | NS/H<br>PS/H | $N$ = 1675<br>1223 | FVC, $FEV_1$<br>FEF 25/75 | Concerning FEF 25/75 in females aged 40 years or more: statistically significant differences between NS and PS/H. Dose–effect relationship between reduction of lung function in PS/H and daily tobacco consumption of the husband. |

* See text for abbreviations.

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never smokers and passive smokers at home with regard to the FEF 25/75 parameter, and also a dose–effect relationship between the reduction in pulmonary function in passive smokers and the daily tobacco consumption of the subject's spouse. However, these relationships were statistically significant only for women, and then only for certain age groups. To date, White and Froeb (35) are the only authors who unreservedly find that passive smoking leads to an impairment of pulmonary function. The predicted value reductions in the case of persons passively exposed to tobacco smoke at the workplace do not, however, differ essentially from those seen in active smokers consuming between 1 and 10 cigarettes a day. This result is difficult to reconcile with a rational dose–effect relationship under the assumptions made earlier [see also Lebowitz (19)].

Taking our own results into consideration, it seems that the passive inhalation of tobacco smoke at home or in the workplace by healthy individuals probably does not lead to any essential impairment of pulmonary function. To what extent this also applies to extreme passive exposure to smoke must remain the subject of further investigation. Thus, for example, persons working in restaurants or in poorly ventilated rooms may be exposed to high levels of smoke pollution.

## ACKNOWLEDGMENTS

We thank Dr. Mentzel, Bundesanstalt für Arbeit, Nuremberg; Dr. Jahn, from the firm of Diehl, Nuremberg; Dr. Bressel and Dr. Heynen from the firm of Siemens, Erlangen, Medical Engineering Group, and the management of the various firms, the personnel directors, and the employee representatives who supported our investigation and made the study possible. Further, we express our special thanks to Mrs. Schmidt and Mrs. Weydanz for carrying out the pulmonary function analyses and establishing the data records.

## REFERENCES

1. Bake, B. Is maximum mid-expiratory flow rate sensitive to small airways obstruction? *Eur. J. Respir. Dis.* 62, 150–151 (1981).
2. Berend, N., Wright, J. L., Thurlbeck, W. M., Martin, G. E., and Woolcock, A. J. Small airways disease: Reproducibility of measurement and correlation with lung function. *Chest* 79, 263–268 (1981).
3. Camner, P., Philipson, K., and Arvidsson, T. Withdrawal of cigarette smoking: A study on tracheobronchial clearance. *Arch. Environ. Health* 26, 90–92 (1973).
4. Cherniack, R. M., and Raber, M. B. Normal standards for ventilatory function using an automated wedge spirometer. *Amer. Rev. Respir. Dis.* 106, 38–46 (1972).
5. Dodge, R. The effects of indoor pollution on Arizona children. *Arch. Environ. Health* 37, 151–155 (1982).
6. Cochrane, G. M., Prieto, F., and Clark, T. J. H. Intrasubject variability of maximal expiratory flow volume curve. *Thorax* 32, 171–176 (1977).
7. Comstock, G. W., Meyer, M. B., Helsing, K. J., and Tockman, M. S. Respiratory effects of household exposures to tobacco smoke and gas cooking. *Amer. Rev. Respir. Dis.* 124, 143–148 (1981).
8. Cosio, M. H., Ghezzo, H., Hogg, J. C., Corbin, R., Loveland, M., Dosman, J., and Macklem, P. T. The relation between structural changes in small airways and pulmonary function tests. *New Engl. J. Med.* 298, 1277–1281 (1977).
9. Cosio, M. G., Hale, K. A., Niewoehner, D. E. Morphologic and morphometric effects of prolonged cigarette smoking on the small airways. *Amer. Rev. Respir. Dis.* 122, 265–271 (1980).
10. Gelb, A. F., Williams, A. J., and Zamel, N. Spirometry—$FEV_1$ vs FEF 25–75 Percent. *Chest* 84, 473–747 (1983).
11. Hasselblatt, U., Humble, C. G., Graham, M. G., and Anderson, H. S. Indoor environmental determinants of lung function in children. *Amer. Rev. Respir. Dis.* 123, 479–85 (1981).

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12. Hegud, C., Hawkins, L. H., and Astrup, P. Exposure of passive smokers to tobacco smoke constituents. *Int. Arch. Occup. Environ. Health* 42, 21–29 (1978).
13. Hegud, C. Indoor air pollution with smoke constituents—An experimental investigation. *Prev. Med.* 13, 582–588 (1984).
14. Kauffmann, F., Tessier, J. F., and Oriol, P. Adult passive smoking in the home environment: A risk factor for chronic airflow limitation. *Amer. J. Epidiol.* 117, 269–280 (1983).
15. Klosterkötter, W., and Gono, E. Zum Problem des Passivrauchens. *Zentralbl. Bakteriol. Hyg. I. Abt. Orig. B* 162, 51–69 (1976).
16. Knudson, R. J., Burrows, B., and Lebowitz, M. D. The maximal expiratory flow-volume curve: Its use in the detection of ventilatory abnormalities in a population study. *Amer. Rev. Respir. Dis.* 114, 871–879 (1976).
17. Knudson, R. J., Slatin, R. C., Lebowitz, M. D., Burrow, B. The maximal expiratory flow-volume curve. Normal standards, variability and effects of age. *Amer. Rev. Respir. Dis.* 113, 587–600 (1976).
18. Lebowitz, M. D., Armet, D. B., and Knudson, R. The effect of passive smoking on pulmonary function in children. *Environ. Internat.* 8, 371–373 (1982).
19. Lebowitz, M. D. "Influence of Passive Smoking on the Pulmonary Function—A Survey." Unpublished data.
20. McFadden, E. R., and Linden, D. A. A reduction in maximum mid-expiratory flow rate. *Amer. J. Med.* 52, 725–737 (1972).
21. McFadden, E. R., Kiker, R., Holmes, B., and Degroot, W. J. Small airway disease. *Amer. J. Med.* 57, 171–182 (1974).
22. McFadden, E. R., and Ingram, R. H. Peripheral airway obstruction. *J. Amer. Med. Assoc.* 235, 259–260 (1976).
23. Morris, J. F., Koski, A., and Johnson, L. C. Spirometric standards for healthy nonsmoking adults. *Amer. Rev. Respir. Dis.* 103, 57–67 (1971).
24. Niewoehner, D. E., Kleinerman, J., and Rice, D. B. Pathologic changes in the peripheral airways of young cigarette smokers. *New Engl. J. Med.* 10, 755–758 (1974).
25. Petty, T. L., Silvers, G. W., Stanford, R. E., Baird, M. A., and Mitchell, R. S. Small airway pathology is related to increased closing capacity and abnormal slope of Phase III in excised human lungs. *Amer. Rev. Respir. Dis.* 121, 449–456 (1980).
26. Quanjer, P. H., (Ed.). "Standardized Lung Function Testing." European Community for Coal and Steel. Luxembourg, July 1983.
27. Schilling, R. S. F., Letai, A. D., Hui, S. L., Beck, G. J., Schoenberg, J. B., and Bouhuys, A. Lung function, respiratory disease and smoking in families. *Amer. J. Epidemiol.* 106, 274–283 (1977).
28. Schulz, V. Small airways disease. *Prax. Pneumol.* 34, 187–218 (1980).
29. Speizer, F. E., Ferris, B. J., Bishop, Y. M. M., and Spengler, J. Respiratory disease rates and pulmonary function in children associated with $NO_2$ exposure. *Amer. Rev. Respir. Dis.* 121, 3–10 (1980).
30. Tager, I. B., Weiss, S. T., Rosner, B., and Speizer, F. E. Effect of parental cigarette smoking on the pulmonary function of children. *Amer. J. Epidemiol.* 110, 15–26 (1979).
31. Thurlbeck, W. M. Smoking, airflow limitation, and the pulmonary circulation. *Amer. Rev. Respir. Dis.* 122, 183–186 (1980).
32. Vutuc, C. Lungenkrebsrisiko und Passivrauchen: Quantitative Überlegungen. *Zentralbl. Bakteriol. Hyg. I. Abt. Orig. B* 177, 90–95 (1983).
33. Weiss, S. T., Tager, I. B., Speizer, F. E., and Rosner, B. Persistent wheeze: Its relationship to respiratory illness, cigarette smoking and level of pulmonary function in a population sample of children. *Amer. Rev. Respir. Dis.* 122, 697–707 (1980).
34. Weiss, S. T., Tager, I. B., Schenker, M., and Speizer, E. The health effects of involuntary smoking. *Amer. Rev. Respir. Dis.* 128, 933–942 (1983).
35. White, J. R., and Frueb, H. F. Small-airways dysfunction in nonsmokers chronically exposed to tobacco smoke. *New Engl. J. Med.* 302, 720–723 (1980).
36. Kruskal, W. H., and Wallis, W. A. Use of ranks in one-criterion variance analysis. *J. Amer. Statist. Assn.* 47, 583–627 (1952).

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Indoor and Ambient Air Quality, 1988.

# PASSIVE TOBACCO SMOKE INHALATION AND LUNG FUNCTION IN ADULTS

M.Kentner & D. Wehle

Institut für Arbeits- und Sozial-Medizin, University of Erlangen-Nuremberg, Schillerstraße 25/29, 8520 Erlangen, FRG

ABSTRACT

To find an answer to the question, whether passive inhalation of tobacco smoke may cause a reduction of lung function, an investigation involving 1,364 white collar workers with healthy lungs was carried out. The group was divided into never smokers, passive smokers, ex-smokers, and current smokers. Lung function was tested by measuring the flow-volume curve standardized for age, height and body weight. Our findings confirmed, as expected, that active smoking causes an impairment of pulmonary function. Ex-smokers and passive smokers showed no flow reductions, which were out of the range of the normal variability. From this it follows that average everyday passive smoke exposure in the office or at home does not lead to essential impairments of lung function in healthy adults.

## INTRODUCTION

There is no doubt that active inhalation of tobacco smoke can cause a reduction in pulmonary function. This applies both to the ventilation with decreased forced expiratory vital capacity (FVC) and to forced expiratory volume in one second ($FEV_1$) (4, 14, 22, 26, 30, 49, 51, 55). Furthermore obstructions of the small airways have been discovered in active smokers by other authors using end-expiratory flows of the flow-volume (MEFV)-curve (4, 9, 16, 29, 37, 44).

Our knowledge of the effects of passive smoking on the bronchopulmonary system, however, is still far from being complete (Surveys in 2, 3, 15, 17, 21, 28, 32, 34, 35, 52, 57).

Studies in children are of particular interest because developing lungs may be more sensitive to the effects of environmental tobacco smoke. Therefore until now already 24 epidemiologic research papers have been published on the effects of current or life-time parental smoking on pulmunary function in children.

Some studies demonstrate a significant negative effect of parental smoking (e.g. 47, 48, 50, 56, 61), other results are inconsistent (e.g. 18, 53, 54) and a number of investigations fail to find any associations between parental smoking habits and a decreased pulmonary function in their children (e.g. 13, 33, 42, 46). These inconsistencies may depend on misclassification bias, socioeconomic status, outdoor air quality, home heating, air conditioning and humidity, genetic factors and in utero or lactational mechanisms by maternal smoking (survey in 62).

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In persons with bronchopulmonary prior diseases, i.e. bronchial hyperreactivity, chronic bronchitis or asthma, passive smoking can either aggravate or lead to a relapse of the illness as is reported in some studies (12, 41, 45, 59). Other authors such as Lebowitz (31), did not find these effects.

In view of these partly contradictory findings the question arises to what extend passive smoking can impair lung function in healthy adults. Only a few studies have dealt with this question and their results are not uniform. To obtain a better insight into this issue, we carried out a study involving a larger group of white collar workers in 1982 - 1983 (27). While our evaluation at that time concentrated on the time-volume curve, our attention is focussed at present on the flow-volume curve.

## PATIENTS AND METHODS

A total of 1,351 subjects (951 men and 413 women) took part in the study. All of them were office workers and participated voluntarily under blind study conditions. The subjects filled out a standardized questionnaire containing among other questions regarding their smoking habits and bronchopulmonary prior diseases.

Subsequently the overall group was divided into four subgroups:

- Never smokers (NS), defined as persons who have never been regularly exposed to tobacco smoke, either actively or passively.
- Passive smokers (PS), defined as subjects who have never actively smoked, but who are currently exposed to passive smoking.
- Ex-smokers (ES), defined as persons who had given up active smoking at least six months previously.
- Current smokers (CS) representing the group of persons who, at the time of the investigation were actively smoking cigarettes, cigars or pipes.

The size of the subgroups, sex ratios, average age and prior diseases in the lower respiratory system are specified in tab. 1.

As statistical analysis of associations between lung function in healthy subjects and persons with prior diseases such as pneumonia, asthma, tuberculosis of the lungs and chronic bronchitis showed no significant differences, subjects with prior illnesses were not excluded.

| | No. of subjects examined | | Age in years (median values) | | Bronchopulmonary prior diseases (pneumonia, asthma, tuberc. of the lungs) | | Chronic bronchitis | |
|---|---|---|---|---|---|---|---|---|
| | M | F | M | F | M | F | M | F |
| NS | 146 | 66 | 42 (31-51) | 41 (33-55) | 21 | 7 | 3 | 2 |
| PS | 252 | 133 | 39 (28-52) | 37 (23-51) | 30 | 18 | 8 | 3 |
| ES | 301 | 60 | 43 (34-55) | 39 (26-49) | 42 | 8 | 9 | 3 |
| CS | 252 | 154 | 40 (30-53) | 31 (22-43) | 34 | 13 | 10 | 8 |
| Su. | 951 | 413 | 41 (31-53) | 35 (26-50) | 127 | 46 | 30 | 16 |

Tab. 1: Number of subjects examined, average age and bronchopulmonary prior diseases in the subgroups.

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Lung functions tests were carried out using an electronic spirometer with a so-called spiroceptor[1]. Test subjects remained in a sitting position for the breathing exercises. The flow-volume curves were recorded by a xy-recorder[2] under BTPS-conditions.

Generally the respiration manoeuvres were repeated two to three times, with only the best curve (plausible figure, maximal values) being used for evaluation.

The following parameters were determined from the flow-volume curve:
- Forced expiratory vital capacity (FVC),
- peak expiratory flow (PEF) with the additional volume (PEF-V),
- maximal expiratory flows at 75, 50, 25 and 15 % of the remaining FVC (MEF 75, MEF 50, MEF 25, MEF 15).

| | | AGE (YEARS) | HEIGHT (M) | WEIGHT (KG) | CONST. |
|---|---|---|---|---|---|
| FVC | M | - 0.026 | 5.766 | - 0.007 | - 3.633 |
| | F | - 0.029 | 4.493 | - 0.008 | - 2.220 |
| PEF | M | - 0.021 | 8.410 | - 0.040 | - 2.221 |
| | F | - 0.057 | 4.234 | 0.041 | - 1.375 |
| PEF-V | M | - 0.008 | 0.157 | - 0.001 | - 0.883 |
| | F | 0.003 | 0.134 | - 0.003 | - 0.541 |
| MEF 25 | M | - 0.021 | 5.854 | - 0.039 | 1.015 |
| | F | - 0.054 | 3.733 | 0.027 | - 0.209 |
| MEF 50 | M | - 0.019 | 4.203 | - 0.023 | - 0.054 |
| | F | - 0.049 | 2.398 | 0.020 | 0.578 |
| MEF 75 | M | - 0.015 | 2.345 | - 0.010 | - 0.973 |
| | F | - 0.031 | 1.890 | - 0.003 | - 0.215 |
| MEF 15 | M | - 0.007 | 1.226 | - 0.005 | - 0.599 |
| | F | - 0.023 | 2.071 | - 0.005 | - 1.418 |

Tab. 2: Correlation factors for adjustment of the pulmonary functio parameters

Normal values were determined for these parameters on the basis of the measurements obtained in the never-smoker group. For this multiple regression analysis was used by adjustment for sex, age, height and weight (tab. 2). The confidence interval of standardized normal values for each subgroup is shown cross-hatched in fig. 1 - 4.

The difference between real and nominal values was analyzed statistically by a matched pairs signed rank test (60). Statistical significances are marked with one ($p = 0.05$) or two asteriks ($p = 0,01$), resp. (fig. 1 - 4).

## RESULTS AND DISCUSSION

### Current smokers

The findings of our study support the thesis, mentioned in the introduction, that active inhalation of tobacco smoke leads to impairments of pulmonary function. A decrease in MEF 50, MEF 25, MEF 15 and FVC was found in male CS. Female CS even showed a degradation in all parameters (fig. 1).

1) Siregnost FD 10, Fa. Siemens, Erlangen, FRG
2) xy-Recorder E 2218, Fa. Siemens, Erlangen, FRG

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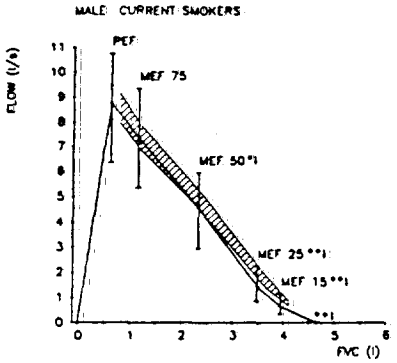



Fig. 1: MEFV-curves in male and female current smokers

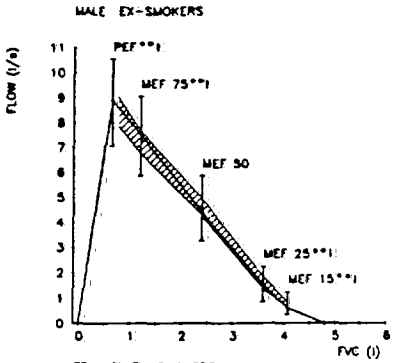
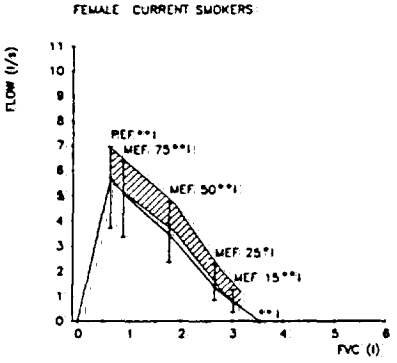
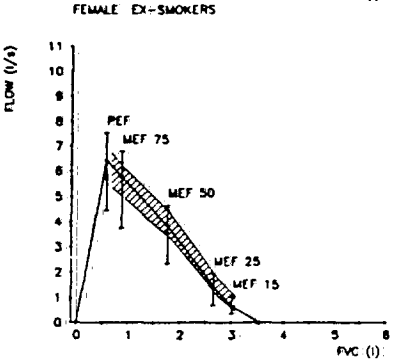



Fig. 2: MEFV-curves in male and female ex-smokers




This means that in men the effect of tobacco smoke inhalation is predominantly reflected in a small airways dysfunction, whereas in women active inhalation shows more severe effects (36). The depression of the entire flow-volume curve in female CS indicates more central bronchial obstructions (25).

The fact that women consume fewer cigarettes than men and also spend a shorter period as smokers shows the particular health hazard in women. While women reach 6.7 packyears (20 cig./day), men have a consumption of 16.6 packyears (tab. 3).

Ex-smokers

We found no essential worsening of lung function in ex-smokers either in men or in women (fig. 2). The only exception here is the decrease in MEF 25 and MEF 15 in men. On the other hand, PEF and MEF 75 in male ES show an increase.

Mean ex-smoker period in men was 7.0 years and in women 4.5 years (tab. 2). Apparently during that time a restitutio ad integrum occurs, also in women, although the impairment of their lung function due to active smoking is more pronounced than in men. Similar results have been obtained

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| | | Cigarettes/day | Smoker years | Pack years | Ex-smoker years |
|---|---|---|---|---|---|
| ES | M | 20.0 (9.0-37.5) | 15.0 (7.5-22.5) | 15.0 (3.5-42.0) | 7.0 (1.5-12.5) |
| | F | 14.0 (5.5-22.5) | 9.5 (2.5-18.0) | 6.3 (0.5-20.5) | 4.5 (1.0- 9.5) |
| CS | M | 19.0 (1.0-29.0) | 17.5 (8.5-29.0) | 16.6 (0.2-42.0) | - |
| | F | 14.5 (6.5-24.0) | 9.5 (4.5-19.0) | 6.7 (1.5-22.8) | - |

Tab. 3: Smoking habits in ex-smokers and current smokers (median values and 66 2/3 confidence intervals).

in intervention studies (5, 7, 8). For example Buist et al. (8), by measuring lung function standard parameters, demonstrated a clear improvement in ventilation after smoking cessation within a period of six to seven months.

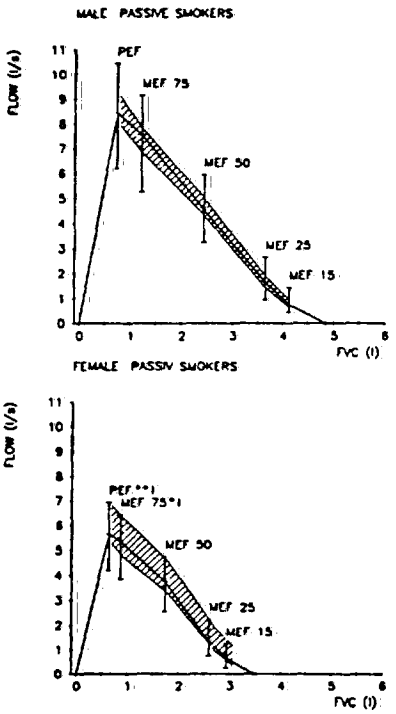


Fig. 3: MEFV-curves in male and female passive smokers

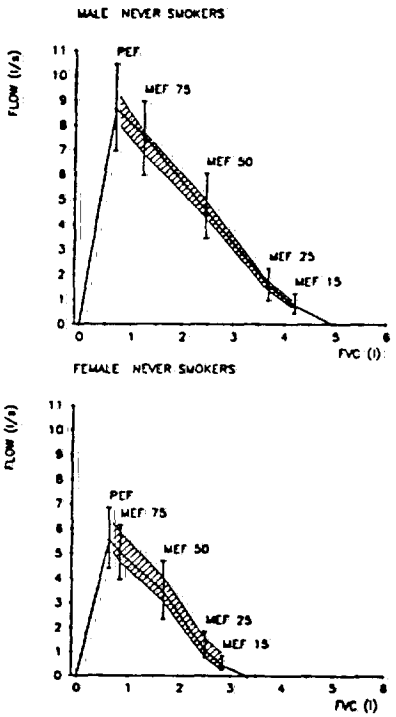


Fig. 4: MEFV-curves in male and female nonsmokers

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Passive smokers

Fig. 3 shows no disturbance in lung function for male passive smokers. In women PEF and MEF 75 still lie within the normal confidence interval, but the statistical analysis reveals a reduction.

This finding is difficult to interpret. If these changes are to be attributed to passive smoke exposure, the results found for active smokers would lead us to expect a decrease in the other flow rates as well. A possible explanation for the isolated reduction of PEF in female PS might be the relatively high interindividual variability of the flow-volume curve. This may lead to accidental correlations.

Never smokers

As the NS group also represents the reference population, no statistically significant alterations of their values can be expected (fig. 4).

Findings by other authors

Up to now eight studies have been published on passive smoking and lung function in healthy adults (tab. 4). These are exclusively cross-section studies.

Only in two studies the flow-volume curves were determined besides recording the time-volume curves. Whilst Brunnekreef et al. (6) found a statistically significant reduction of PEF and MEF 75 in PS in comparison to NS, Schilling et al. (42) were unable to establish such a relationship for any lung function parameter.

The remaining six studies which concentrated only on the time-volume curve did not show uniform results. Whereas three investigations confirm a reduction of lung function due to passive smoking (23, 38, 58), this relationship was not supported by the other three studies (11, 19, 27).

These investigations differ considerably in the methods applied. This discrepancies are mainly found in formation of subgroups, lung function pro-

| AUTHORS | SUBGROUPS (n)* | PULMONARY FUNCTION PARAMETERS | RESULTS |
|---|---|---|---|
| SCHILLING ET AL. (1977) (42) | n NS, F NS (138); n NS, F CS (40); n CS, F NS (74); n CS, F CS (78) | FVC, $FEV_1$, PEF, MEF 50, MEF 25 | NO STATISTICALLY SIGNIFICANT REDUCTION OF LUNG FUNCTION IN PS/N. |
| WHITE AND FROEB (1980) (58) | n NS (200), F NS (200); n PS/N (200), F PS/N (200); n CS (650), F CS (650) | FVC, $FEV_1$, PEF 25-75, FEF 75-85 | FEF 25-75 AND FEF 75-85 IN MF PS/N SHOW STATISTICALLY SIGNIFICANT LOWER VALUES THAN IN NS. |
| COMSTOCK ET AL. (1981) (11) | n PS/N (369), F PS/N (49); n ES (179), F ES (17); n CS (472), F CS (142) | $FEV_1$, $FEV_1$/FVC | NO STATISTICALLY SIGNIFICANT REDUCTION OF LUNG FUNCTION IN MF PS/N. |
| HELSING ET AL. (1982) (19) | MF NS (372); MF PS/N (189) | $FEV_1$, $FEV_1$/FVC | THE PRESENCE OF POOR VENTILATORY FUNCTION IN PS/N SHOWED ONLY A VERY WEAK ASSOCIATION (N.S.) |
| PETROVIC ET AL. (1982) (38) | n NS/N, PS/N (434); F NS/N, PS/N (741) | $FEV_1$/VC | STATISTICALLY SIGNIFICANT DIFFERENCES BETWEEN THE GROUPS (EXACT STUDY DESIGN WAS NOT AVAILABLE). |
| KAUFFMANN ET AL. (1983) (23) | n NS (849), F NS (826); n PS/N (65), F PS/N (1158) | FVC, $FEV_1$, PEF 25-75 | IN THE COMPARISON OF NS AND PS/N AGED 40 YEARS OR MORE STATISTICALLY SIGNIFICANT DECREASE APPEARED IN WOMEN FOR $FEV_1$ AND FEF 25-75. |
| KENTNER ET AL. (1984) (27) | n NS (142), F NS (66); n PS/N (39), U (146), UU (36); F PS/N (18), U (61), UU (36); n CS (301), F ES (59); n CS (247), F CS (153) | FVC, PEF 25-75, FEF 75-85 | NO STATISTICALLY SIGNIFICANT REDUCTION OF LUNG FUNCTION IN MF PS/N, U, UU. |
| BRUNNEKREEF ET AL. (1985) (6) | F NS (30, 36); F PS/N (27, 39) | FVC, $FEV_1$, FVC, PEF, MEF 75, MEF 50, MEF 25, MMEF | PEF AND MEF 75 WERE STATISTICALLY SIGNIFICANT DECREASED IN F PS/N. |

* N = AT HOME; U = AT WORKPLACE.
n = MALE; F = FEMALE.
OTHER ABBREVIATIONS SEE TEXT.

Tab.4: Review of literature published so far with regard to the topic pulmonary function in healthy adult passive smokers

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cedures, standardization and, moreover, age structure of the participants. Therefore, any direct comparison can only be limited.

The best way to compare them is by calculating the differences in lung function parameters between NS or normal persons on the one hand and in PS on the other. The data available can be seen in tab. 5. In female PS flow decreases for PEF and MEF 75 between 10 to 16 % and 9 to 14 %, resp. were found. For both male and female PS, decreases in FEF 25 - 75 and FEF 75 - 85 can be calculated which are between 6 and 9 % or 5 and 15 %, resp.

Reductions of this magnitude in our investigations were only found in comparing with NS and CS. Thus in female CS PEF was decreased by 19 % and MEF 75 by 14 %. For FEF 75 - 85 reductions were between 8 and 18 % in CS (27).

| | | | | |
|---|---|---|---|---|
| BRUNNEKREEF ET AL. (1985) | | | | |
| - NS vs. PS/H (AT PRESENT EXPOSED AGED 40 - 60 YEARS, ONLY WOMEN) | F PEF | • | 0.75 L/s | (10 %) |
| | F MEF 75 | • | 0.58 L/s | (9 %) |
| - NS vs. PS/H (EXPOSED SINCE ABOUT 15 YEARS. AGED 40 - 60 YEARS , ONLY WOMEN) | F PEF | • | 1.33 L/s | (16 %) |
| | F MEF 75 | • | 0.97 L/s | (14 %) |
| WHITE & FROEB (1980) | | | | |
| - NORMAL PERSONS VS. PS/H | M FEF 25-75 | • | 0.20 L/s | ( 9 %) |
| | F FEF 25-75 | • | 0.19 L/s | ( 7 %) |
| | M FEF 75-85 | • | 0.05 L/s | ( 5 %) |
| | F FEF 75-85 | • | 0.12 L/s | (15 %) |
| KAUFFMANN ET AL. (1985) | | | | |
| - NS vs. PS/H (AGED 40 YEARS OR MORE) | F FVC | • | 0.09 L/s | ( 3 %) |
| | F $FEV_1$ | • | 0.09 L/s | ( 4 %) |
| | F FEF 25-75 | • | 0.17 L/s | ( 6 %) |

Tab. 5: Differences of lung function parameters in non-smokers with statistically significant association between passive smoke exposure and lung function reduction.

This would mean that the strain put upon lung function impaired by passive smoking is comparable with that by active tobacco smoke inhalation. That this cannot in fact be the case is emphasized by estimating the doses of the most principal bronchial irritants found in tobacco smoke to which the passive smoker is exposed. At sedentary or easy physical work over an 8-hour workshift cigarette equivalents of between 0.04 and 8 cigarettes may be taken into account, depending on the substances measured (tab. 6). Especially with regard to the equivalent of 8 cigarettes concerning nitrogen oxides it has to be assumed that other sources than tobacco smoke have not been taken into account. The substantial deviation is due to the great variability of the external exposure. This depends primarily on the amount of tobacco smoke emission and the ventilation of the room (39) while the internal exposure is mainly determined by the ventilations of the lungs.

Taking cigarette equivalents into consideration, the effects of passive smoking on lung function found by White & Froeb (58), Kauffmann et al. (23) and Brunnekreef et al. (6) are too high. A positive confounder in these investigations might be that persons having pre-existing impairments of lung function or bronchial hyperreactivity never take up smoking (51, 55) and so are over-represented in the PS group.

Furthermore, the question arises to what extent the described impairments of lung function due to passive smoking are of pathophysiological relevance. In 1984, the American Thoracic Society (1) published a diagram

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| | |
|---|---|
| PARTICULATE MATTER | |
| - RESPIRABLE PARTICULATES | 0.0% - 0.2% |
| VOLATILE SUBSTANCES | |
| - NITROGEN OXIDES | CA. 8 |
| - ACROLEIN | 0.80 - 4.00 |
| - FORMALDEHYDE | ? |
| - AMMONIA | ? |

Tab. 6: Cigarette equivalents for passive smokers concerning respiratory membrane irritants (Henschler 1983)

showing the various degrees of damages to the respiratory system from air pollution, which demonstrates a wide transitional field between physiological and pathophysiological alterations (fig. 5). This applies particularly to lung function parameters (24).

In general it may be assumed that under normal everyday conditions passive smoke exposure does not induce relevant pathological changes of bronchopulmonary function in healthy adults in the sense of adverse effects.

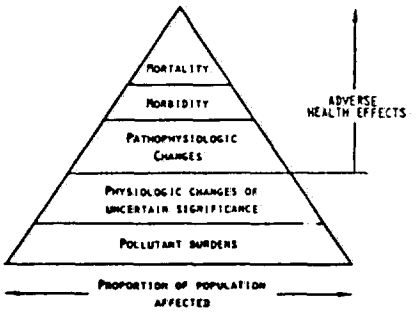


Fig. 5: Spectrum of biological response to pollutant exposure (American Thoracic Society 1984)

## CONCLUSIONS

From the findings available so far there is no evidence that average everyday passive smoke exposure in the office or at home leads to an essential reduction of lung function in healthy adults.

Under unfavourable conditions (forced and passive smoke exposure over many years, small rooms, heavy physical work, prior illnesses of the respiratory system) a reduction in lung function due to passive smoking might be possible, particularly in women.

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## REFERENCES


1. American Thoracic Society (1984) Am. Thorac. Soc. News 10, 6-8.
2. Bake, B. (1983) in 40.
3. Bake, B. (1984) Eur. J. Resp. Dis. 65 (suppl. 133), 85-87.
4. Beck, G.J., Doyle, C.A., Schachter, E.N. (1981) Am. Rev. Resp. Dis. 123, 149-155.
5. Bosse, R., Sparrow, D., Rose, C.L., Weiss, S.T. (1983) Am. Rev. Resp. Dis. 123, 378-381.
6. Brunnekreef, B., Fischer, P., Remijn, B., Van der Lende, R., Schouten, J., Quanjer, P. (1985) Int. J. Epidemiol. 14, 227-230.
7. Buist, A.S., Sexton, G.J., Nagy, J.M., Ross, B.B. (1976) Am. Rev. Resp. Dis. 114, 115-122.
8. Buist, A.S., Nagy, J.N., Sexton, G.J. (1979) Am. Rev. Resp. Dis. 120, 953-957.
9. Cochrane, G.M., Prieto, F., Hickey, B., Benatar, S.R., Clark, T.J.H. (1974) Thorax 29, 389-394.
10. Cochrane, G.M., Prieto, F., Clark T.J.H. (1977) Thorax 32, 171-176.
11. Comstock, G.W., Meyer, M.B., Helsing, K.J., Tockman, M.S. (1981) Am. Rev. Resp. Dis. 124, 143-148.
12. Dahms, T.E., Bolin, J.E., Slavin, R.G. (1981) Chest 80, 530-534.
13. Dodge, R. (1982) Arch. Environ. Health 37, 151-155.
14. Ferris, B.G., Anderson, D.O., Zickmantel, R. (1965) Am. Rev. Resp. Dis. 91, 252-261.
15. Gelband, H., Larson, B., Kronebusch, K., U.S. Congress, May 1986.
16. Greitenberg, B. (1985) Prax. Klin. Pneumol. 39, 165-169.
17. Guyatt, G.H., Newhouse, M.T. (1985) Chest 88, 445-451.
18. Hasselbladt, U., Humble C.G., Graham, M.G., Anderson, H.S. (1981) Am. Rev. Resp. Dis. 123, 479-85.
19. Helsing, K.J., Comstock, G.W., Mayer, M.B., Tockman, M.L. (1982) Environ. Int. 8, 365-370.
20. Henschler, D. (ed.), VCH Verlagsgesellschaft, Weinheim (FRG) 1985.
21. Holt, P.G., Turner, K.J. (1983) in 40.
22. Jancik, E.H., Hansma, T.W., Janssen, W., Schouten, J.P., Tiesinga-van der Veen, T., Jansen-Santema, T. (1980) Prax. Pneumol. 34, 657-680.
23. Kauffmann, F., Tessier, J.-F., Oriol, P. (1983) Am. J. Epidemiol. 117, 269-280.
24. Kentner, M. (1987) ASP-Schriftenreihe, Bd. 73, Gentner, Stuttgart.
25. Kentner, M. (1987) Atemw.- und Lungenkrh. 13, 485-490.
26. Kentner, M., Weltle, D. (1985) in: Verhandlungen der Deutschen Gesellschaft für Arbeitsmedizin, Dortmund; Gentner, Stuttgart.
27. Kentner, M., Triebig, G., Weltle, D. (1984) Prev. Med. 13, 656-669.
28. Klosterkötter, W., Gono, E. (1976) Zbl. Bakt., Hyg., I. Abt. Orig. B. 162, 51-69.
29. Knudson, R.J., Slatin, R.C., Lebowitz, M.D., Burrows, B. (1976) Am. Rev. Resp. Dis. 113, 587-600.
30. Knudson, R.J., Bloom, J.W., Knudson, D.E., Kaltenborn, W.T. (1984) Chest 86, 20-29.
31. Lebowitz, M.D. (1983) in 40.
32. Lebowitz, M.D. (1984) Prev. Med. 13, 645-655.
33. Lebowitz, M.D., Armet, D.B., Knudson, R. (1982) Environ. Int. 8, 371-373.
34. Lee, P.N. (1982) Fd. Chem. Toxic. 20, 223-229.
35. Lefcoe, N.M., Ashley, M.J., Pederson, L.L., Keays, J.J. (1983) Chest 84, 90-95.
36. Macklem, P.T., Thurbleck, W.M., Fraser, R.G. (1971) Ann. Int. Med. 74, 167-175.
37. Peters, J.M., Ferris, B.G. (1967) Am. Rev. Resp. Dis. 95, 774-782.


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38. Petrovic, B., Petrovic, O., Slavkovic, V., Stevanovic, S. (1982) World Conference of the International Union against Tuberculosis, Buenos Aires, April 21-24, 1982.
39. Repace, J.L., Lowrey, A.H. (1982) Ashrae Transactions 88, 895-914.
40. Rylander, R., Peterson, Y., Snella, M.-C. (eds.) ETS-Environmental Tobacco Smoke. University of Genena, Switzerland 1983.
41. Savel, F. (1970) Arch. Environ. Health 21, 146-148.
42. Schilling, R.S.F., Letai, A.D., Hirs, S.L., Beck, S.T., Schoenberg, J.B., Bouhuys, A. (1977) Am. J. Epidemiol. 106, 274-283.
43. Schulz, V. (1980) Prax. Pneumol. 34, 187-218.
44. Seely, J.E., Zuskin, E., Bouhuys, A. (1971) Sciences 14, 741-743.
45. Shephard, R.J., Collins, R., Silverman, F. (1979) Environ. Res. 20, 392-402.
46. Speizer, F.E., Ferris, B.J., Bishop, Y.M.M., Spenger, J. (1980) Am. Rev. Resp. Dis. 121, 3-10.
47. Tager, I.B., Weiss, S.T., Rosner, B., Speizer, F.E. (1979) Am. J. Epidemiol. 110, 15-26.
48. Tager, I.B., Munoz, A., Rosner, B., Weiss, S.T., Carey, V., Speizer, F.E. (1985) Am. Rev. Resp. Dis. 131, 752-759.
49. Tashkin, D.P., Clark, V.A., Coulson, A.H., Bourque, L.B., Simmons, M., Reems, C., Detels, R., Rokaw, S. (1983) Am. Rev. Resp. Dis. 128, 12-16.
50. Tashkin, D.P., Clark, V.A., Simmons, M., Reems, C., Coulson, A.H., Bourque, L.B. et al. (1984) Am. Rev. Resp. Dis. 129, 891-897.
51. Tockman, M., Menkes, H., Cohen, B. (1976) Am. Rev. Resp. Dis. 114, 711-722.
52. Valentin, H., Bost, H.-P., Wawra, E. (1978) Zbl. Bak. Hyg., I. Abt. Orig. B 167, 405-434.
53. Vedal, S., Schanker, M.B., Samet, J.M., Speizer, F.E. (1984) Am. Rev. Resp. Dis. 130, 187-192.
54. Ware, J.H., Dockery, D.W., Spiro, A., Speizer, F.E., Ferris, B.G. (1984) Am. Rev. Resp. Dis. 129, 366-374.
55. Webster, P.M., Corimer, E.G., Man, S.F.P., Woolf, C.R., Zamel, N. (1979) Am. Rev. Resp. Dis. 119, 223-228.
56. Weiss, S.T., Tager, I.B., Speizer, F.E., Rosner, B. (1980) Am. Rev. Resp. Dis. 122, 697-707.
57. Weiss, S.T., Tager, I.B., Schenker, M., Speizer, F.E. (1983) Am. Rev. Resp. Dis. 128, 933-942.
58. White, J.R., Froeb, H.F. (1980) N. Engl. J. Med. 302, 720-723.
59. Wiedemann, H.P., Mahler, D.A., Loke, J., Virgulto, J.A., Snyder, P., Matthay, R.A. (1986) Chest 89, 180-185.
60. Wilcoxon, F., Katti, S.K., Wilcox, R.A. (1963) Lederle Laboratories, Division Amer. Cyanamid Company, Pearl River, New York 1963.
61. Yarnell, J.W.J., St. Leger, A.S. (1979) Br. J. Dis. Chest 73, 230-236.
62. Witorsch, R.J., Witorsch, P. (1987) (not published paper until now)

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# Psychophysiological Response to Environmental Tobacco Smoke in an Experimental Social Setting

G. Winneke, M. Neuf, A. Roscovanu, and H.-W. Schlipköter

## Summary

Acute psychophysiological effects of environmental tobacco smoke (ETS), corresponding to carbon monoxide (CO) concentrations of 5 and 10 ppm and produced by an active smoker, were studied in 72 adult nonsmokers and compared to the effects of traffic noise (65 and 75 dB(A)) and of odorexposure (50 and 150 ppb of hydrogen sulfide). This sample represents the first block out of 126 subjects (Ss) for which data analysis was completed by November 1987. Experiments took place in an actively ventilated exposure chamber in a social setting. Objective measures, namely, heart rate, blood pressure, rate of respiration, eye blinks, and lacrymal flow as well as subjective measures of annoyance based on questionnaire items were taken four times during exposure; carboxyhemoglobin (COHb) was measured before and after exposure.

Whereas cardiorespiratory variables were not affected by ETS exposure, COHb, eyeblinks as well as part of the annoyance-data exhibited ETS-induced elevation, although not necessarily at the 5 ppm-level in each case. ETS-induced annoyance at 5 and 10 ppm dCO was comparable to noise-induced annoyance at 65 and 75 dB(A), respectively. Ss preclassified as either strongly or weakly annoyed by environmental conditions in their neighbourhoods (e.g. traffic noise or industrial odors), as assessed in a social survey covering 2,300 individuals, also did exhibit significant differential susceptibility in terms of ETS-induced annoyance. From these still preliminary findings the tentative conclusion is drawn for a no-adverse-effect-level (NOEL) for acute psychophysiological effects of ETS to be located near the 5 ppm-level in terms of dCO, and for annoyance to represent a more generalized, stable reaction-tendency rather than a reaction to a specific environmental condition.

## Introduction

Most previous studies dealing with acute psychophysiological effects of environmental tobacco smoke (ETS) have used smoking machines to produce desired levels of sidestream smoke exposure (Weber et al. 1976, 1979, 1981; Muramatsu et al. 1983). In deviating from this typical procedure, which is consistent but lacks ecological validity, we conducted a pilot experiment, in which nonsmoking students were exposed to ETS by an active smoker in a social, experimentally controlled setting (Winneke et al. 1984). Objective measures, namely eyeblinks, lacrymal flow, blood pressure, heart rate and respiration, as well as subjective measures of annoyance based on questionnaire-items, were taken four times during exposure; in addition COHb-concentrations were measured before and after ETS-exosure. Significant effects of ETS-exposure were found for COHb, blinks, lacrymal flow, as well as for most of the questionnaire-based information. With

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the exception of some questionnaire-variables, exhibiting dose response-relationship, the majority of the dependent variables showed threshold-characteristics: The 15 ppm-condition only deviated significantly from the control level. From this finding the tentative conclusion was drawn for a no adverse effect-level for acute psychophysiological effects of ETS to be located between 5 and 15 ppm dCO.

In order (1) to substantiate this conclusion, (2) to compare acute psychophysiological effects of ETS to those elicited by noise- and odor-exposure, and (3) to gain information about the more specific or more generalized nature of the annoyance-response, the present extended study was conducted. The experimental part of this study, based on 126 Ss altogether, is finished whereas data-analysis is still preliminary. Partial results based on the first set of 72 Ss are given and discussed here.

## Material and Methods

### *External Conditions*

The experiments took place in a non-climatized but actively ventilated chamber with dimensions (l × w × h) 238 × 177 × 230 cm (9.7 cbm). Air exchange, as calculated from the measured dilution of H2S in the chamber (see below) was 80 cbm/h. Two experimental subjects (Ss) and the informed experimenter who, in case of ETS-exposure, was the active smoker, were sitting in the chamber around a table playing games (Memory). Temperature and humidity in the chamber increased during the experiment, namely from about 57 to 66% (humidity) and from about 18 to 20°C on the average, irrespective of the experimental condition.

### *Experimental Conditions*

During the experiment Ss were exposed in three counterbalanced sessions to ETS, traffic noise and odor, but at only one out of three levels of exposure. In accordance with previous work (e.g. Muramatsu et al. 1983) carbon monoxide (CO) was measured as a convenient, non-specific marker of ETS- exposure by means of non-dispersive infrared (IR-)-spectroscopy (URAS III, Hartmann & Braun, Frankfurt). CO-levels, defined as dCO by taking pre-exposure levels into account, were <1, 5 and 10 ppm, respectively. Active smoking of 0, 4.2 and 7.2 cigarettes of the same brand was necessary on the average to reach these CO-levels.

Traffic noise was presented from calibratedtape-recordings at continuous equivalent sound levels of 65 and 75 dB(A), respectively; background sound pressure-level in the experimental chamber was 60 dB(A). Spot measurements were taken at random intervals by means of a portable sound level-meter (RC 345, Reten-Elektronic, Idstein).

Odor-exposure was done by controlled metering prediluted hydrogen sulfide (H2S) into the chamber from a pressure tank containing a calibrated gas-concentration of 100 ppm. Chamber-concentrations were set at 50 and 150 ppb and were monitored by means of the photometric molybdenum-blue method (VDI, 1974) after about 30 min sampling-time. The odor threshold of H2S is between 1 and 3 ppb; 50 and 150 ppb correspond to perceived intensities of "distinct/strong" and "strong/very strong" according to a six-point rating-scale (Winneke et al. 1979).

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### *Dependent Variables*

Before and after each experiment capillary blood-samples were taken from a fingertip for the determination of COHb by means of gaschromatography after methane conversion; CO-concentrations were converted to COHb by means of average Hb-concentrations for males and females, respectively. At the beginning and at three consecutive intervals during the experiment (15, 35 and 55 min) systolic and diastolic blood-pressure was taken by means of an electronic device (boso digital Standard). In addition the following measures were taken continuously during the experiment but evaluated at the four regular intervals given above for two minutes duration only: Eyeblinks by means of amplified potentials recorded from one eye by means of AgCl-electrodes, rate of respiration by means of thermistor-elements attached to the nose, and heart-rate by means of photo-electric couplers attached to one ear-lobe.

In addition to these objective measures an annoyance-questionnaire was given four times during the experiment, which consisted of 39 five-point statements covering descriptive (e.g. "The air is clean"), symptom-related (e.g. "My eyes are burning"), and emotional-reactive items ("I feel relaxed"). Response-categories ranged from "Don't agree at all" to "Fully agree", or from "Not at all" to "very strong". Part of the items were specific to each of the three conditions (ETS, noise, odor), whereas others covered all three of them. This was particularly true for the emotional-reactive items, as well as for an eleven-point annoyance thermometer with response-categories ranging from 0 (not at all disturbing) to 10 (unbearably disturbing).

Subjects: 72 nonsmokers of both sexes participated. 55% of them were females, and the mean age was 44.6 years with extremes ranging from 18 to 74 years. Four Ss with initial COHb exceeding 5% were treated as suspected smokers and excluded from further analyses, thus leaving 68 Ss for final statistical evaluation. Assignment of Ss to control (N = 23), medium (N = 23), and high (N = 22) intensities of exposure was random.

### *Statistical Procedures*

Both descriptive and inferential statistics were applied. Questionnaire-data were tentatively treated as being of interval-quality. Analysis of variance (mixed model) was used for the testing of main- and interaction-effects (Winer 1962). Significance-levels of 5% and lower were taken to separate significant from non-significant effects.

## Results

### *Objective Information*

CO-uptake during ETS-exposure, as given by COHb-levels, did exhibit the expected alterations (Table 1). Whereas there was a slight, insignificant decrease of COHb for control-Ss during an experimental session on the average, ETS-induced, dose-dependent increase occurred during exposure ($F = 17.3$; $p < 0.0001$). The values given in the table correspond roughly to those of the pilot-study for comparable exposures (Winneke et al. 1984), but are well below limits of biological significance (Winneke 1978).

Cardio-respiratory variables, namely blood-pressure, heart rate and rate of respiration, did not exhibit significant ETS-induced changes (Table 2). There is, however, a tendency for respiration to become slower during exposure at the highest ETS-level.

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Table 1. ETS-related COHb%. Means, standard deviations (SD) and ranges are given

| Groups | Before exposure | | | After exposure | | | |
|---|---|---|---|---|---|---|---|
| | x̄ | (SD) | range | x̄ | (SD) | range | N[a] |
| Control | 0.71 | (0.23) | 0.58–0.84 | 0.68 | (0.19) | 0.58–0.79 | 14 |
| ETS 5 ppm | 0.75 | (0.21) | 0.66–0.84 | 0.87 | (0.19) | 0.79–0.96 | 23 |
| ETS 10 ppm | 0.84 | (0.23) | 0.74–0.94 | 1.03 | (0.22) | 0.93–1.13 | 22 |

[a] COHb-analysis of blood taken after the experiment was done in only part of randomly selected control-Ss.

Table 2. Cardio-respiratory variables. Means (x̄) and standard deviations (SD) are given. 0, 5 and 10 correspond to levels of ETS-exposure in terms of CO

| | CO | 0′ | | 15′ | | 35′ | | 55′ | |
|---|---|---|---|---|---|---|---|---|---|
| | | x̄ | SD | x̄ | SD | x̄ | SD | x̄ | SD |
| Heart-rate | 0 | 78.9 | ( 8.3) | 79.4 | ( 8.5) | 79.0 | ( 8.3) | 78.5 | ( 9.0) |
| | 5 | 78.1 | ( 8.8) | 78.8 | ( 9.3) | 77.2 | ( 8.6) | 76.9 | ( 7.8) |
| | 10 | 78.0 | (10.4) | 78.3 | (10.2) | 76.8 | (10.3) | 77.8 | (11.0) |
| Rate of respirat. | 0 | 18.7 | ( 2.7) | 18.2 | ( 2.7) | 18.6 | ( 2.9) | 18.1 | ( 3.3) |
| | 5 | 17.0 | ( 3.4) | 17.6 | ( 2.8) | 17.1 | ( 2.9) | 17.4 | ( 2.5) |
| | 10 | 17.7 | ( 3.7) | 17.0 | ( 3.9) | 16.5 | ( 3.9) | 15.6 | ( 3.6) |
| Blood-pressure (systol.) | 0 | 117.1 | (14.6) | 114.0 | (13.8) | 113.2 | (15.6) | 115.2 | (19.7) |
| | 5 | 130.9 | (20.3) | 128.7 | (20.1) | 129.2 | (21.6) | 128.1 | (20.3) |
| | 10 | 118.4 | (21.4) | 119.5 | (24.4) | 120.8 | (28.1) | 119.2 | (25.9) |
| Blood-pressure (diast.) | 0 | 116.9 | (18.2) | 111.9 | (16.7) | 111.4 | (20.1) | 114.1 | (16.9) |
| | 5 | 133.1 | (20.6) | 127.9 | (18.6) | 131.3 | (18.6) | 127.2 | (21.8) |
| | 10 | 121.6 | (24.3) | 118.2 | (26.9) | 116.9 | (27.8) | 117.9 | (27.2) |

The number of eyeblinks/minute increased during a one hour experimental session both under control- and exposure-conditions (Fig. 1). Comparatively, however, the rate of increase was faster at the high level of ETS-exposure than at control-level. The overall group- differences are not significant ($F = 1.92$; $p = 0.16$), although, as can be seen from the confidence-limits, the last three sampling-points of the 10 ppm-condition are significantly ($p < 0.05$) elevated. The slight elevation of the 5 ppm-curve is not significant for this sample size.

*Subjective Information*

Descriptive statements (e.g. "The air is clean") exhibited strong ETS-related change; typical examples are given in Fig. 2. These effects were, of course, highly significant ($p <$

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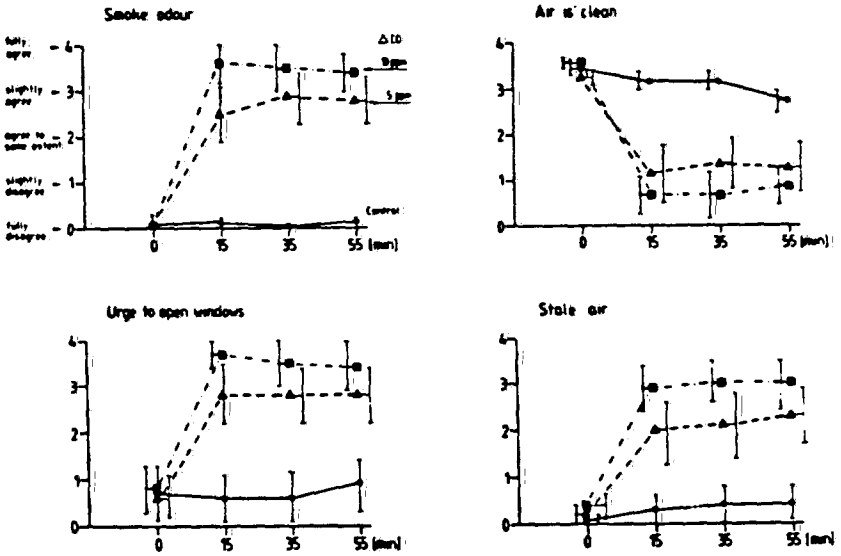


Fig. 1. Results from questionnaire-items describing the experimental situation for different levels of ETS-exposure. Means and confidence-limits (CL-95) are given

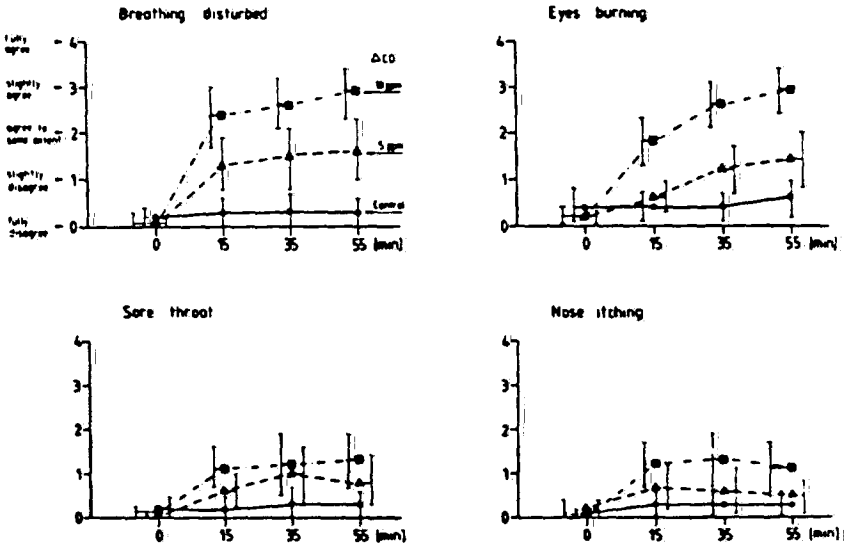


Fig. 2. Results from questionnaire-items related to perceived symptoms during different levels of ETS-exposure. Means and CL-95 are given

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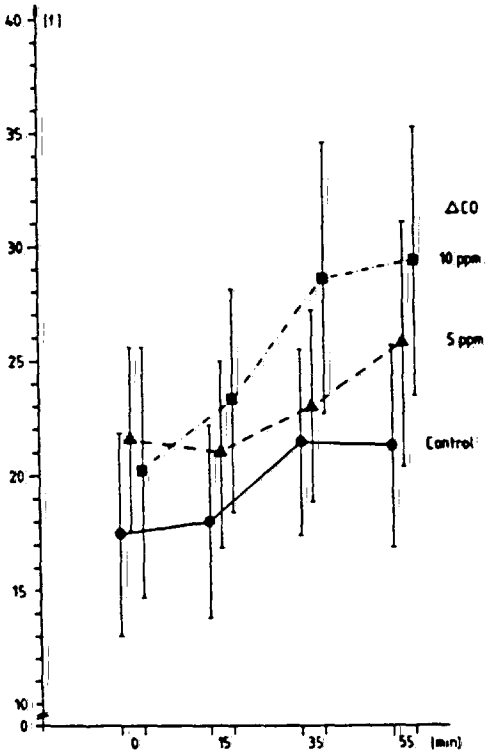


Fig. 3. Number of eyeblinks/minute during exposure for different ETS-levels. Means and CL-95 are given

Table 3. Symptom-related questionnaire-items. F- and p-values characterize the outcome of analysis of variance

| | Main effect | | Interaction | |
|---|---|---|---|---|
| | F | p | F | p |
| Burning eyes | 19.45 | 0.000 | 20.03 | 0.000 |
| Impaired breathing | 19.50 | 0.000 | 14.90 | 0.000 |
| Sore throat | 3.43 | 0.040 | 3.84 | 0.001 |
| Itching nose | 4.52 | 0.015 | 2.65 | 0.017 |
| Headache | 1.17 | 0.310 | 2.96 | 0.009 |
| Dry mouth | 1.87 | 0.170 | 2.15 | 0.049 |

0.0001), both in terms of main effects (intensity of ETS-exposure) and interactions (intensity × time).

Typical examples for symptom-related effects are given in Fig. 3. The statistical results from analyses of variance for these as well as similar statements are shown in Table 3.

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**Table 4.** Emotional-reactive questionnaire-items. F- and p-values characterize the outcome of analysis of variance

| | Main effect | | Interaction | |
|---|---|---|---|---|
| | F | p | F | p |
| Uncomfortable | 9.26 | 0.000 | 7.50 | 0.000 |
| Angry | 3.93 | 0.024 | 3.42 | 0.003 |
| Nervous | 2.19 | 0.120 | 1.80 | 0.100 |
| Cheerful | 2.19 | 0.120 | 0.64 | 0.695 |
| Relaxed | 1.19 | 0.312 | 0.62 | 0.713 |
| Merry | 1.10 | 0.339 | 2.21 | 0.044 |
| Concerned | 0.87 | 0.424 | 1.07 | 0.382 |

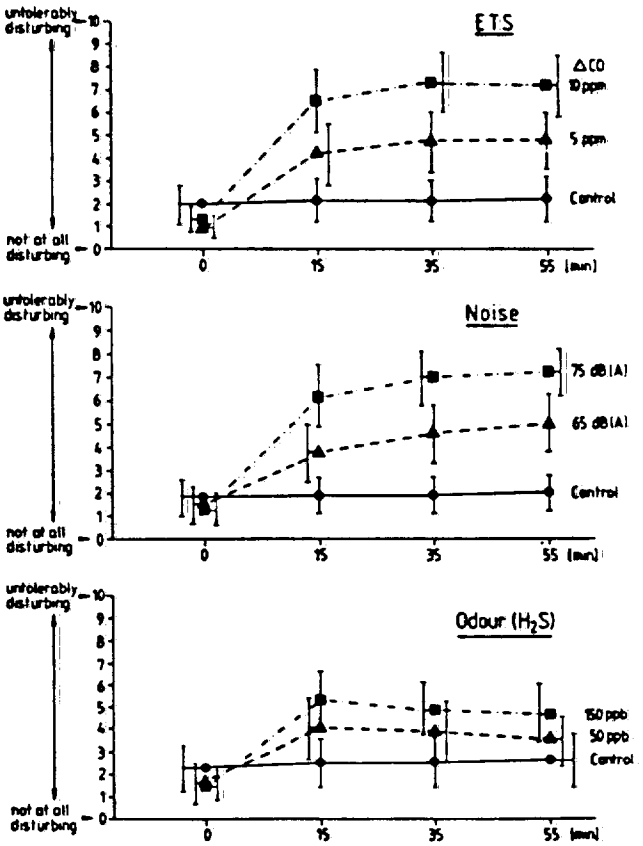


**Fig. 4.** Degree of annoyance during exposure to three levels each of ETS, traffic noise and hydrogen sulfide-odor. Means and CL-95 are given

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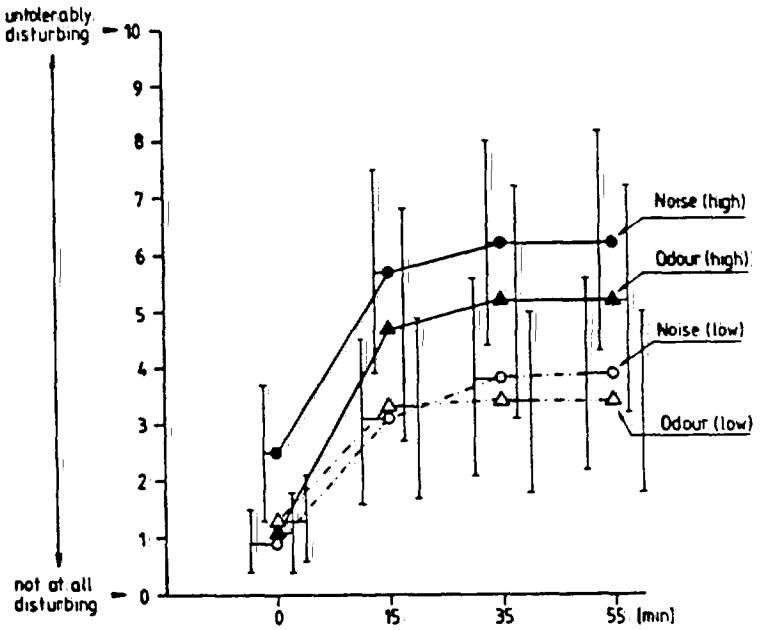


Fig. 5. Degree of annoyance during ETS-exposure for groups of Ss preclassified in terms of either high (upper curves) or low (lower curves) annoyance to environmental noise or odors, respectively. Means and CIl-95 are given

Four out of eight items exhibit significant main effects (intensity), whereas for all of them significant interactions (intensity of exposure × time) were found as well. This means that the time-course is different for control- and exposure-conditions, respectively.

Only two out of seven emotional-reactive statements, namely those expressing feelings of discomfort ($F = 9.26$; $p = 0.000$) and of anger ($F = 3.93$; $p = 0.02$) did show significant main effects associated with levels of ETS-exposure, whereas additional time × exposure-interactions occurred for feelings of discomfort, anger and cheerfulness (Table 4).

For the purpose of comparing the annoying effects of ETS-exposure to that induced by either traffic noise- or odor-exposure data were taken from the eleven point annoyance-scale (see above). The results from that comparison are given in Fig. 4. These curves clearly show that for each of the three conditions highly significant ($p < 0.001$) exposure-related effects occurred which, except for the odor-condition, did exhibit pronounced dose-response-characteristics. It is interesting to note, furthermore, that the degree of annoyance produced by the two ETS-levels is roughly equivalent to continuous sound pressure-levels of 65 and 75 dB(A), respectively, whereas the degree of odor-annoyance induced by hydrogen sulfide is comparatively reduced. This, most likely, can be ascribed to olfactory fatigue or adaptation.

In order to ascertain if Ss, preclassified as either highly or weakly annoyed by either environmental noise or industrial odors in their respective neighbourhoods, these

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extreme groups were compared for degree of annoyance across exposure-levels for the three experimental conditions. Ss preclassified as highly annoyed scored higher in terms of annoyance during ETS-exposure than did Ss preclassified as weakly annoyed (Fig. 5). This group-difference is statistically significant for ETS ($F = 2.80$; $p < 0.05$), but only borderline ($p < 0.12$) for either experimental noise- or odor-exposure. Perceived intensities at the different levels of exposure did not exhibit any group-differences, whatsoever ($0.95 > p < 0.55$).

## Discussion

Carbon monoxide (CO) was used here and in our previous study (Winneke et al. 1984) to characterize the degree of ETS-exposure. Both because of its lack of specificity as well as its inconsistent temporo-spatial associations with other ETS-components CO cannot be considered a truely representative marker of ETS under real-life-conditions (Ball et al. 1987), particularly if, in addition to cigarettes, pipes and cigars contribute to ETS (Klus et al. 1987). No general agreement has, therefore, been reached so far as to which components or component-combinations could be used in order to characterize ETS-exposure in natural settings.

CO, on the other hand, has been shown to display consistent association with suspended particulates (TSP), both in decay-situations as well as in experimental steady-state- or dynamic conditions, in which both the number of cigarettes as well as the rate of air-exchange were varied (Leaderer et al. 1984). In addition correlation between CO-levels on the one hand and number of cigarettes as well as several gas-phase components of ETS, such as NO, aldehydes, acrolein, HCN or formaldehyde, have been reported both for extreme (Hugod et al. 1978) as well as for more moderated degrees of experimental ETS-exposure (Weber et al. 1976).

In the light of this evidence, and in order to be able to compare the outcome of our experiment with that of similar studies (Hugod et al. 1978; Muramatsu et al. 1983; Weber et al. 1976, 1979, 1981; Winneke et al. 1984) in a quantitative manner, the measurement of CO to characterize degrees of ETS-exposure in our experimental setting can be considered a reasonable compromise between describing the true complexity of ETS-exposure on the one hand and relying simply on the number of cigarettes smoked per unit time and volume on the other.

In taking CO as the basis for comparison our cardiovascular findings are consistent with those of others (Weber et al. 1976; Harke and Bleichert 1972), who, at even higher levels of ETS-exposure did not observe exposure-related increase of either heart-rate or blood-pressure. Nicotine-intake at such ETS-levels is likely to be too low for cardio-vascular changes to be expected.

As for eyeblinks our data are somewhat at variance with those of Muramatsu et al. (1983), who describe significant elevation at ETS-levels below 3 ppm dCO, whereas both in our previous (Winneke et al. 1984) and in the present study no significant increase was observed at ETS-levels corresponding to 5 ppm dCO. It is still uncertain as to whether methodological differences can explain these different outcomes. Whereas Muramatsu et al. (1983) relied on intraindividual comparisons based on actual counting of videotaped blinks by observers at predetermined intervals during exposure, our findings are based on interindividual comparisons of a smaller number of Ss by means of continuous electrophysiological recordings of spontaneous blinking. Although the latter technique would generally be considered as having a higher ecological validity, final analysis of the full set of data will be needed to clarify, if

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significant elevation of eyeblinks can in fact be expected to occur at ETS-levels at or even below 5 ppm dCO.

The most clearcut findings of the present study are those related to questionnaire-based annoyance. Descriptive and, to a lesser extent, symptom-related complaints display dose-related increase with some effects already significant at the lowest ETS-level. This was not true in our previous study. Part of this differential outcome is most likely due to the fact that in the present study control-conditions were improved by forced air-exchange. From these still preliminary findings the tentative conclusion is drawn for a non-adverse-effect-level (NOEL) for acute psychophysiological effects of ETS to be located near the 5 ppm-level in terms of dCO.

The present study is the first to compare ETS-induced annoyance – reactions to those associated with either noise- or malodor-exposure. The data presented here strongly suggest that such a comparative approach is feasible, and that ETS-exposure corresponding to dCO-levels of 5 and 10 ppm are equivalent to traffic-noise at sound pressure-levels of 65 and 75 dB(A), respectively. Comparison with odor-annoyance is less convincing because adaptation strongly attenuates perceived odor-intensity for hydrogen sulfide.

Another interesting finding of the present study relates to the observation that preclassification of nonsmoking Ss for environmental annoyance is related to the degree of annoyance under experimental exposure-conditions as well: Ss preclassified as being either strongly or weakly annoyed by environmental odor- or noise-exposure in their respective neighbourhoods, do exhibit strong or weak degrees of annoyance under conditions of ETS-exposure as well. From this the tentative conclusion is drawn for annoyance-responses to be more generalized reaction-tendencies rather than responses elicited by specific environmental conditions.

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## *References*

Ball M, Intorp M, Schilling B (1987) Measurements of ETS-components in indoor air under real life conditions. Int. Conference on "Indoor Air Quality", November 4–6, 1987 in Tokyo

Harke B, Bleichert A (1972) Zum Problem des Passivrauchens. Int Arch Arbeitsmed 29:312–322

Hugod C, Hawkins LH, Astrup P (1978) Exposure of passive smokers to tobacco smoke constituents. Int Arch Occup Environ Health 42:21–29

Klus H, Begutter H, Nowak A, Seits N, Ultsch I (1987) Measurements of ETS-components in indoor air under controlled conditions. Int. Conference on "Indoor Air Quality", November 4–6, 1987 in Tokyo

Leaderer BP, Cain WS, Isseroff R, Berglund LG (1984) Ventilations requirements in buildings – II. Particulate matter and carbon monoxide from cigarette smoking. Atm Environ 18:99–106

Muramatsu T, Weber A, Muramatsu S, Akermann R (1983) An experimental study on irritation and annoyance due to passive smoking. Int Arch Occup Environ Health 51:305–317

Verein Deutscher Ingenieure = VDI (1974) Messung gasförmiger Immissionen. Messen von Schwefelwasserstoff-Konzentrationen. Molybdän-blau-Sorptionsverfahren. VDI 2454. VDI-Verlag, Düsseldorf

Weber A (1981) Verunreinigung der Raumluft durch Tabakrauch: Grundlagen zur Beurteilung der Luftqualität und der Lüftung. Habilitationsschrift. ETH Zürich

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Weber A, Fischer T, Grandjean E (1976) Objektive und subjektive physiologische Wirkungen des Passivrauchens. Int Arch Occup Environ Health 37:277–288

Weber A, Fischer T, Grandjean E (1979) Passive smoking in experimental and field conditions. Environ Res 20:205–216

Winer BJ (1962) Principles in experimental design. McGraw-Hill, New York San Francisco Toronto London

Winneke G (1978) Grenzwertüberlegungen für Kohlenmonoxid. In: Ges. z. Förderung d. Lufthygienefoschung (ed) Med. Inst. für Lufthygiene und Silikoseforschung, Jahresbericht 1977. Giradet, Essen, 75–90

Winneke G, Kotalik J, Keldenich HO, Kastka J (1979) Zur Wahrnehmung von Schwefelwasserstoff unter Labor- und Feldbedingungen. Staub-Reinhalt Luft 39: 156–159

Winneke G, Plischke K, Roscovanu A, Schlipköter HW (1984) Patterns and determinants of reaction to tobacco smoke in an experimental exposure setting. In: Berglund B, Lindvall T, Sundell J (eds) Indoor air, vol 2: Radon, passive smoking, particulates and housing epidemiology. Swedish Council for Building Research, Stockholm, pp 351–356

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Health and Welfare Canada
Santé et Bien-être social Canada

The Occupational Health and Safety

Department of the Human Resources Division

Ottawa Civic Hospital

October 24, 1985

# SICK BUILDING SYNDROME:

# CAUSES AND EFFECTS

John Kirkbride, M.B., M.Sc.
Director
Occupational Health Unit
Medical Services Branch
Health and Welfare Canada
du Chardon Street
Tunney's Pasture
OTTAWA, Ontario
K1A 0L3

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SICK BUILDING SYNDROME: CAUSES AND EFFECTS

Sick Building Syndrome can be broken down into three sub-categories - Tight Building Syndrome, Humidifier Fever and Legionnaire Disease. TABLE 1.

I will spend most of my time this morning on Tight Building Syndrome.

What is a Tight Building? The definition is arbitrary, practical, and loose. TABLE 2.

There are buildings which meet these criteria in which one can see through gaps between walls and window frames. Paradoxically, some buildings with tight building envelopes - energy efficient residences, for example - do not meet the criteria, since the occupants are able to control the ventilation system.

The health effects that make up Tight Building Syndrome: TABLE 3.

Few researchers would disagree with this list. However, there may be some debate as to whether these are health effects or comfort factors. Is eye irritation an illness? Is headache that clears up on leaving the building an illness? Is fatigue an illness? One can argue either case. The point is not entirely academic, since in real life employees are generally asked to contact health service staff about health problems, and building maintenance staff about comfort problems ...

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You will also note that most of the listed symptoms are rather vague and subjective, and therefore very difficult to measure in a consistent way. Vague and subjective the symptoms may be, yet the syndrome is striking and convincing, and a time-consuming problem to those of us who have responsibilities in the fields of occupational and environmental health. You will also note that a number of these symptoms are attributable to tobacco smoke, a near universal contaminant of office buildings. Until the last few years, tobacco smoke has been looked upon as a normal inoccuous component of indoor air. I will return to tobacco smoke, and some other contaminants, shortly.

Many organizations in many countries have investigated Tight Building Syndrome. The problem was first described in Scandinavia in the early nineteen seventies. By 1975, it was being frequently described in North America.

An American agency that has conducted large numbers of fairly detailed investigations is NIOSH (the National Institute of Occupational Safety and Health). Health and Welfare Canada experience is similar to that of NIOSH.

TABLE 4.
TABLE 5.

The causes of Tight Building Syndrome

TABLE 6.

First, let me mention a non-cause: lack of oxygen. I can say with confidence that the oxygen level in this building and in virtually every office building in Canada

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today is 21%. My organization no longer measures oxygen levels when investigating office buildings with air quality problems.

TABLE 7.

TABLE 8.

TABLE 9.

Pollutants

Let me review some of the pollutants that may be contributory; and let me say in advance that in practice the search for pollutants is usually - not always - unrewarding. With one notable exception: tobacco smoke. Most investigators and researchers will agree that this is the most important single pollutant in the air of office buildings.

Carbon monoxide

Major indoor sources (unvented gas stoves, heaters, etc.) are not usually a problem in office buildings. Motor vehicle emissions can sometimes enter a building - from an attached parking garage, for example.

CO generated by cigarettes would not usually result in indoor levels exceeding industrial standards, although this can occur in small meeting rooms containing numerous smokers.

Formaldehyde

Measured levels in office buildings infrequently exceed the Canadian residential standard of 0.1 ppm. Formaldehyde is a common component of several types of

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Source: https://www.industrydocuments.ucsf.edu/docs/zqyx0000

condensation-type synthetic resins (e.g. phenol formaldehyde, melamine-formaldehyde, urea formaldehyde.) Urea formaldehyde resin is used in plywood (adhesive) and particle board (binder). Coated paper, floor coverings and fabrics can contains formaldehyde.

Carbon dioxide

Product of combustion and human metabolism. Outdoor levels typically 320 ppm. Office levels typically 600 ppm, depending on quality of ventilation and density of occupants. $CO_2$ is measured as a proxy of the quality of ventilation.

Ozone

A power oxidizer, and reactive pollutant. The dominant source is the outdoor air, formed by photochemical reaction. Indoor sources are unusual e.g. electrostatic air cleaners, electric motors, photocopiers.

Ventilation factors as a cause of Tight Building Syndrome

From a previous slide (Slide 5), you will remember that in the experience of NIOSH, in about 50% of the buildings investigated, the problems were attributed to faulty ventilation, temperature, and humidity. Let us look at some standards for temperature and humidity:

These standards may be met for a floor of a building, yet there may still be local problems, created for example by room dividers which restrict airflow.

Temperature and humidity problems are relatively common.

TABLE 10.

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Source: https://www.industrydocuments.ucsf.edu/docs/zqyx0000

Psychological factors

These can be very important.

The occupant is aware that he is the best sensor in a building - better able to integrate environmental information than thermometers or hygrometers or airflow meters. Yet he does not have the control over the ventilation system that these instruments may have. The only method left open to him is to express his dissatisfaction to the people who have access to the controls. If these complaints do not produce improvement, the occupant feels out of control, and can become increasingly irritated by these conditions and increasingly sensitive to them. We must also bear in mind that ventilation systems are increasingly complex, and that in a large building only the designer may fully understand them. In other words, maintenance staff may not be in full control of the system.

HUMIDIFIER FEVER

Humidifier fever is an allergic illness. The picture is that of a mild flu' like illness - chills, fever, breathlessness, typically on Mondays on returning to work.

Humidifer fever is a type of extrinsic allergic alveolitis - comparable to farmers lung.

The cause: microorganisms in the humidifying system - bacteria, fungi, protozoa and thermophilic actinomycetes. TABLE 11.

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Source: https://www.industrydocuments.ucsf.edu/docs/zqyx0000

LEGIONNAIRES DISEASE

You may remember the episode of 1976, at the Bellevue Stratford Hotel in Philadelphia. There were 182 cases of pneumonia-like illness, including 29 deaths, in participants at the annual convention of the American Legion. The diagnosis was elusive. Sabotage, nickel carbonyl poisoning, swine flu, were all suspected, then rejected. Eighteen months later the Centres for Disease Control in the USA isolated and identified Legionella pneumophilia (a bacterium). (The final solution at the Bellevue Stratford hotel was truly final: the hotel was demolished.)

Spread of Legionella in humans: by aerosol from contaminated water systems (e.g. shower heads). Person to person spread has not been described.

How frequent is Legionnaires Disease?

In the USA, in the first four months of 1985, 190 cases were notified. This compares with 102 cases of typhoid, or 982 cases of measles during the same time period.

Other aspects of Legionella are disturbing:

TABLE 12.
TABLE 13.

... /7

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LUNG CANCER

This disease is important enough to merit discussion in the context of building associated illness.

When I reviewed some building pollutants, I included radon, asbestos, and secondary cigarette smoke. These agents are all human carcinogens, and can cause lung cancer as well as some other cancers. TABLE 14.

Radon is generally present in much lower levels in office buildings than in residences. Therefore I will not consider it further.

Asbestos is often encountered in building materials, and I would be surprised if this building was an exception. I would also predict that if we were to attempt to measure asbestos in the air of this room it would not be in present in detectable quantities. Asbestos does not present a significant health problem in buildings unless it is damaged, allowing fibres to enter the air.

With regard to secondary tobacco smoke, the evidence from a number of epidemiological studies has been steadily accumulating since 1979. The weight of scientific evidence strongly suggests that secondary tobacco smoke in the workplace is responsible for a significant number of lung cancers - somewhere between 300 and 500 lung cancer deaths per year in Canada.

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In conclusion, let me summarize the causes and effects of Sick Building Syndrome: TABLE 15.

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# TABLES

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Source: https://www.industrydocuments.ucsf.edu/docs/zqyx0000

# SICK BUILDING SYNDROME

1. TIGHT BUILDING SYNDROME

2. HUMIDIFIER FEVER
   (AN INFLUENZA - LIKE ILLNESS)

3. LEGIONNAIRES DISEASE

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Source: https://www.industrydocuments.ucsf.edu/docs/zqyx0000

## DEFINITION OF TIGHT BUILDING

LARGE BUILDING

FORCED VENTILATION

OCCUPANTS CANNOT OPEN WINDOWS OR CONTROL OWN VENTILATION

USUALLY NEW OR NEWLY RENOVATED BUILDING

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Source: https://www.industrydocuments.ucsf.edu/docs/zqyx0000

## HEALTH EFFECTS OF TIGHT BUILDING SYNDROME

EYE IRRITATION

HEADACHE

ODOR

SKIN IRRITATION/RASH

SINUS CONGESTION

COUGH

SORE THROAT

SHORTNESS OF BREATH

ABNORMAL TASTE

DIZZINESS

FATIGUE

NAUSEA

WHEEZING AND HYPERSENSITIVITY

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Source: https://www.industrydocuments.ucsf.edu/docs/zqyx0000

## PRESUMED SOURCE OF THE PROBLEM IN 203 INVESTIGATIONS BY NIOSH

| | Number | % |
|---|---|---|
| Poor Ventilation, Thermal Comfort, or Humidity | 107 | 53 |
| Contaminants from Inside the Building (Copiers, Tobacco Smoke, etc.) | 42 | 21 |
| Contaminants from Outside the Building (Motor Vehicle Exhaust, etc.) | 21 | 10 |
| Building Fabric Contamination (fibreglass, formaldehyde, glues, etc.) | 7 | 3 |
| Biological Contamination | 7 | 3 |
| Miscellaneous or Undetermined | 19 | 10 |
| | 203 | |

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Source: https://www.industrydocuments.ucsf.edu/docs/zqyx0000

# INDOOR AIR QUALITY INVESTIGATIONS
# MEDICAL SERVICES BRANCH
# 1984

| Problem Type Found | Building Investigations | |
|---|---|---|
| | Number | % |
| 1. Inadequate Ventilation | 64 | 68 |
| - poor air circulation | | |
| - inadequate outdoor air ($CO_2$ > 800 ppm) | | |
| - poor temperature/ humidity control | | |
| 2. Outdoor Contaminant | 9 | 10 |
| - reentry of building exhaust | | |
| - motor vehicle exhaust | | |
| 3. Indoor Contaminant | 5 | 5 |
| - copy machines | | |
| - tobacco smoke | | |
| 4. Building Fabric | 2 | 2 |
| - glues and adhesives | | |
| - formaldehyde and organics | | |
| 5. Biological Contaminants | 0 | 0 |
| 6. No problem identified | 14 | 15 |
| TOTAL: | 94 | 100 |

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Source: https://www.industrydocuments.ucsf.edu/docs/zqyx0000

## CAUSES OF TIGHT BUILDING SYNDROME

1. POLLUTANTS

2. VENTILATION FACTORS:

   - TEMPERATURE
   - HUMIDITY
   - AIRFLOW

3. PSYCHOLOGICAL FACTORS

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Source: https://www.industrydocuments.ucsf.edu/docs/zqyx0000

## POLLUTANTS

### Carbon monoxide

Typical office levels: 0.5 - 5 ppm

Industrial standard: 35 - 50 ppm

Ambient air standard: 9 ppm

### Formaldehyde

UFFI rare in federal office buildings

More important sources:

Carpet backing, fabrics, insulation, particle board;

small amounts from tobacco smoke

Typical office levels: less than 0.1 ppm

Residential standard (Canada): 0.1 ppm

Industrial standard: 1 ppm

### Radon

Not a problem in office buildings

Does not give rise to acute symptoms

### Asbestos

Not usually a problem unless damaged or disturbed

Does not give rise to acute symptoms

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Source: https://www.industrydocuments.ucsf.edu/docs/zqyx0000

Carbon dioxide

Typical outdoor levels: about 330 ppm

Typical indoor levels: about 600 - 1000 ppm

Industrial standard: 5000 ppm

Ozone

Typical outdoor level: 0 - 40 ppb

Typical indoor level: 0 - 20 ppb

Industrial standard: 100 ppb

Viable organisms (bacteria, fungal spores, amoebae, protozoa, nematodes)

No standards

Odors

Standard: acceptability to panel of observers.

Negative ions

No acceptable scientific evidence to suggest that they affect comfort or health.

Photochemical smog

An ingenious hypothesis

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Source: https://www.industrydocuments.ucsf.edu/docs/zqyx0000

## MOST FREQUENT OFFENDERS

TOBACCO SMOKE

CARBON MONOXIDE

FORMALDEHYDE

ODORS

VIABLE ORGANISMS?

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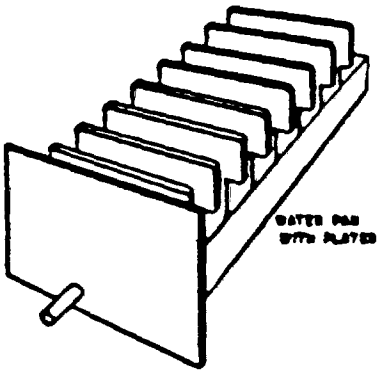
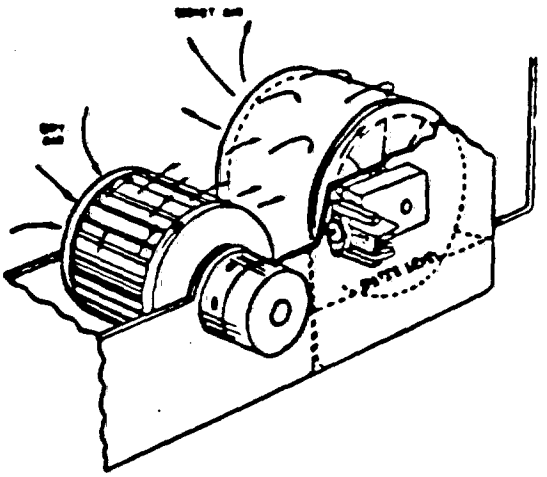
Source: https://www.industrydocuments.ucsf.edu/docs/zqyx0000

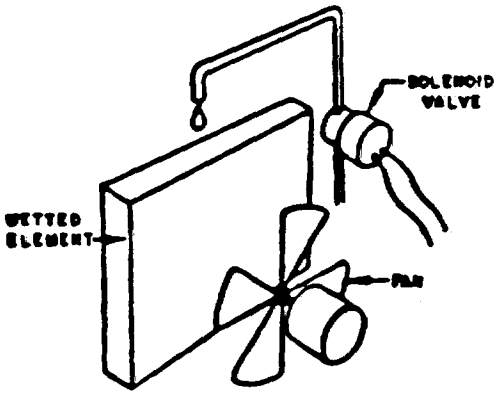
## HUMIDIFIERS



Pan-type Humidifier



Wetted Drum-type Humidifier




Power-type Wetted Element Humidifier

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Source: https://www.industrydocuments.ucsf.edu/docs/zqyx0000

## PUBLIC WORKS CANADA STANDARDS

| | |
|---|---|
| ACCEPTABLE INDOOR TEMPERATURE RANGE | 19°C - 26°C |
| ACCEPTABLE HUMIDITY RANGE | 20% - 60% |

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Source: https://www.industrydocuments.ucsf.edu/docs/zqyx0000

## A QUÉBEC CITY HOSPITAL, 1983

12 CASES OF LEGIONNAIRES DISEASE OCCURRED IN HOSPITAL (NOSOCOMIAL INFECTION)

WATER SAMPLES FROM 38 FAUCETS (IN ROOMS):

30% POSITIVE FOR LEGIONELLA

HOT WATER TANKS:

1 OUT OF 2 POSITIVE

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Source: https://www.industrydocuments.ucsf.edu/docs/zqyx0000

## QUÉBEC CITY, 1983

54 DOMESTIC WATER HEATERS WERE EXAMINED

ELECTRIC WATER HEATERS: 11 OUT OF 37 POSITIVE FOR LEGIONELLA

OIL OR GAS WATER HEATERS: 0 OUT OF 17 POSITIVE FOR LEGIONELLA

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Source: https://www.industrydocuments.ucsf.edu/docs/zqyx0000

## LUNG CANCER

~~RADON~~

ASBESTOS

SECONDARY TOBACCO SMOKE

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Source: https://www.industrydocuments.ucsf.edu/docs/zqyx0000

# SICK BUILDING SYNDROME: SUMMARY

## EFFECTS

SICK BUILDING SYNDROME ENCOMPASSES: TIGHT BUILDING SYNDROME
HUMIDIFIER FEVER
LEGIONNAIRES DISEASE

## CAUSES

POLLUTANTS - CHEMICAL (INCLUDING SOME CARCINOGENS)
- BIOLOGICAL AGENTS

VENTILATION, TEMPERATURE, HUMIDITY PROBLEMS

PSYCHOLOGICAL FACTORS

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Source: https://www.industrydocuments.ucsf.edu/docs/zqyx0000

# BIBLIOGRAPHY

2023383272

Source: https://www.industrydocuments.ucsf.edu/docs/zqyx0000

BIBLIOGRAPHY


Ager, B.P., and Tickner, J.A. "The Control of Micro-Biological Hazards Associated with Air-Conditioning and Ventilation Systems." Annals of Occupational Hygiene, 1983, 27, 341-358.

American Conference of Governmental Industrial Hygienists Documentation of the Threshold Limit Values for Substances in the Workroom Air. 4th Edition. Cincinnati: ACGIH, 1980.

American National Standards Institute, and American Society of Heating, Refrigerating and Air-Conditioning Engineers. ANSI/ASHRAE Standard 62-1981. Ventilation for Acceptable Indoor Air Quality. New York: American Society of Heating, Refrigerating and Air Conditioning Engineers, Inc., 1980.

Arnow, Paul M., et al. "Early detection of Hypersensitivity Pneumonitis in Office Workers." American Journal of Medicine, 1978, 64, 236-242.

ASHRAE Standard. Ventilation for Acceptable Indoor Air Quality. The American Society of Heating, Refrigerating, and Air-Conditioning Engineers, Inc., 1791 Tullie Circle, N.E., Atlanta, GA 30329, ANSI/ ASHRAE 62, 1981, 1-20.

ASHRAE Standard. Thermal Environmental Conditions for Human Occupancy. The American Society of Heating, Refrigerating, and Air-Conditioning Engineers, Inc., 1791 Tullie Circle, N.E., Atlanta, GA 30329, ASHRAE 55, 1981, 1-17.

Cain, W.S. and Leaderer, B.P. "Ventilation Requirements in Occupied Spaces during Smoking and Non-Smoking Occupancy." Environment International, 1982, 8, 505-514.

Canada Diseases Weekly Report. Nosocomial Legionnaires' Disease in the Québec City Area, November 3, 1984, 10-44, 173-174.

Canada Diseases Weekly Report. Legionella and Domestic Hot Water Heaters in the Québec City Area, November 3, 1984, 10-44, 175.

Canada Diseases Weekly Report. Legionella Pneumophila Infections, Scotland, 1983. November 3, 1984, 10-44, 175-176


... /2

20233837/8

Source: https://www.industrydocuments.ucsf.edu/docs/zqyx0000

Carlton-Foss, J.A. "The Tight Building Syndrome." ASHRAE Journal, December 1983, 25, 38-41.

Colligan, M.J. "Psychological Effects of Indoor Air Pollution." New York Academy of Medicine. Bulletin, 1981, 57, 1014-1026.

Couch, R.B. "Viruses and Indoor Air Pollution." New York Academy of Medicine. Bulletin, 1981, 57, 907-921.

Huber, G. and Wanner, H.U. Indoor Air Quality and Minimum Ventilation Rate. Environment International, 1983, 9, 153-156.

Int-Hout, D. and Berger, P. (Members ASHRAE) What's Really Wrong with VAV Systems. ASHRAE Journal, December, 1984, 36-38.

McDonald, J. Corbett. Investigation of Employee Health Complaints at Les Terrasses de la Chaudière. Final Report to TB/PSAC Steering Committee, Treasury Board of Canada Contract TB/CT-REQ B8059, July 1984, 1-41.

McGregor, R.G., Vasudev, P., Létourneau, E.G., McCullough, R.S., Prantl, F.A. and Taniguchi, H. Background Concentrations of Radon and Radon Daughters in Canadian Homes. Health Physics, August, 1980, 39, 285-289.

Moghissi, A.A., Moghissi, B.D. Environment International. A Journal of Science, Technology, Health, Monitoring and Policy. Pergamon Press, 1982, 8, (1-6), 1-534.

Moschandreas, D. "Indoor Air Quality Relationship - A Critical Review." Journal of the Air Pollution Control Association, September 1982, 32, 904-907.

National Academy of Sciences Committee on Indoor Pollutants. Indoor Pollutants. National Academy Press, Washington, D.C., 1981.

National Primary and Secondary Ambient Air Quality Standards. Environmental Protection Agency. Federal Register, Thursday, February 8, 1979, 44, 28, 8202-8221.

Photochemical Oxidants; Measurement of Ozone in the Atmosphere; Requirements for Preparation, Adoption, and Submittal of Implementation Plans. Environmental Protection Agency. Federal Register, Thursday, June 2, 1978, 43, 121, 26972-26981

Proceedings of the 3rd International Conference on Indoor Air Quality and Climate. Held in Stockholm, August 20-24, 1984, 23-29.

... /3

20233879

Source: https://www.industrydocuments.ucsf.edu/docs/zqyx0000

"Sick Building Syndrome - The Search for a Cure." Occupational Hazards, 1983, 45, 85-86.

Space Environmental Standards. Preface to the 1979 Edition. Standards and Guidelines Mechanical Design. Public Works Canada, 1979-01-02.

Spengler, J.D. et al. "Indoor Air Pollution: a Public Health Perspective." Science, July 1983, 221, 9-17.

Sterling, E. and Sterling, T. "The Impact of Different Ventilation Levels and Fluorescent Lighting Types on Building Illness: An Experimental Study." Canadian Journal of Public Health, November-December, 1983, 74, 385-392.

Sterling, Theodor D. "Economics and Politics in the Assessment of Causes of Building Illness: the NAS/NRC Report on Indoor Pollutants." International Journal of Health Services, 1984, 14, 43-53.

Stolwijk, Ian A.J. "Tight Building Syndrome." Toxic Substances Journal, Winter 1983/84, 5, 155-161.

Robert, S., Bernstein, A.D., William, G., Sorenson, A. et al. Exposures to Respirable, Airborne Penicillium from a Contaminated Ventilation System: Clinical, Environmental and Epidemiological Aspects. Am. Ind. Hyg. Assoc. J., 1983, 44(3), 161-169

Turiel, I., Hollowell, C.D., Miksch, R.R., Rudy, J.V. and Young R.A. The Effects of Reduced Ventilation on Indoor Air Quality in an Office Building. Atmospheric Environment, 1983, 17, 1, 51-64.

Turiel, I. and Rudy, J. Occupant-Generated $CO_2$ as an Indicator of Ventilation Rate. Lawrence Berkeley Laboratory, University of California, Energy and Environment Division, LBL-95d, EEB-Vent 80-15, Prepared for the U.S. Department of Energy under Contract W-7405-ENG-48, 1-20.

Walkinshaw, Douglas S. Indoor Air Quality Research in Canada. Prepared for Presentation at the Ener-Health '84 Conference, Winnipeg, Manitoba, October 16-17, 1984, Based on NRC/DBR publication, NRCC 23775. 1-20.

Wallingford, Kenneth M. NIOSH Indoor Air Quality Investigations in Non-Industrial Workplaces: An Update. Cincinnati: National Institute for Occupational Safety and Health, 1984.

... /4

20233783780

Source: https://www.industrydocuments.ucsf.edu/docs/zqyx0000

Walsh, P.J., Dudney, C.S. and Copenhaver, E.D. Indoor Air Quality. CRC Press, Inc., 1984, 1-207.

Woods, J.E. "Ventilation, Health and Energy Consumption: A Status Report." ASHRAE Journal, 1979, 21(7), 23-27.

World Health Organization, Regional Office for Europe, Copenhagen. Indoor Air Pollutants: Exposure and Health Effects. Report on a WHO meeting. EURO Reports and Studies 78. World Health Organization, 1983, 1-42, ISBN 92 890 1244 7.

Yocom, John E. Indoor-Outdoor Air Quality Relationships. A Critical Review, Journal of the Air Pollution Control Association, Copyright 1982, 500-920.

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Source: https://www.industrydocuments.ucsf.edu/docs/zqyx0000

# Indoor air quality — the NIOSH experience

JAMES MELIUS, M.D., KENNETH WALLINGFORD, M.S., RICHARD KEENLYSIDE, M.D., and JAMES CARPENTER
National Institute for Occupational Safety and Health

## Introduction

Health complaints associated with poor indoor air quality have been reported with increasing frequency among office workers over the past 10 years. A number of factors have contributed to this trend, including changes in building design to increase energy efficiency, introduction of new building materials, and increased awareness among workers of potentially toxic exposures in their work and home environments. While some exposures in the indoor environment may have serious implications for the health of the occupants (e.g., radon daughters, asbestos, formaldehyde, pathogenic microorganisms, allergens), the health risks of most indoor air exposures are poorly understood. Nevertheless, office workers who are concerned about a possible problem in their work environment often demand a thorough investigation of the environment and of its potential for health risks. For the investigator, this is commonly a challenging but frustrating experience because of the lack of guidelines and evaluation criteria for the "non-industrial" setting.

The health complaints reported by the occupants of the typical "problem building" are usually diverse and non-specific, and rarely point to an obvious cause. Also, the nature of the problem may be obscured by reports of a variety of serious, but unrelated, medical conditions associated with the office environment.

Appropriate environmental sampling is often quite difficult because of the presence of low levels of many ubiquitous substances. Specific contaminants from sources in the office can be readily measured and quantitated (e.g., ozone from copying machines), but more often very low levels of a variety of chemicals are detected. Such sampling results may in one sense be reassuring, but they have limited usefulness when linked with nonspecific symptoms. These investigations are often complicated by extensive media coverage, multiple earlier investigations by other groups, and deteriorating labor relations. The "Federal expert" often comes into the picture when, despite much basic work having been done, the situation has reached a "crisis."

This paper will briefly present and review the indoor air quality investigations conducted by the National Institute for Occupational Safety and Health (NIOSH) since the start of the Health Hazard Evaluation Program. These investigations are being presented not as "THE WAY" to evaluate such problems, but, rather, to review our experience and share our insights as we have evolved our approach to these investigations.

## Indoor air quality health hazard evaluations

Through December 1983, NIOSH has completed 203 Health Hazard Evaluations involving indoor air quality (IAQ) in a variety of settings (Table I). (This does not include our investigations of asbestos-related problems in office buildings.) Prior to 1978, only six IAQ evaluations were performed; however, since then, the number has increased dramatically. It appears that in the last 2 years, the number of these completed investigations has leveled off, but this change may reflect our handling of many IAQ inquiries by providing written materials and phone consultation, and by the increased capability of state and local health de-

TABLE I
Completed NIOSH Indoor Air Quality Investigations by Year (through December 1983)*

| Year | Number Completed | % |
|---|---|---|
| Pre-1978 | 6 | 3.0 |
| 1978 | 9 | 4.4 |
| 1979 | 12 | 5.9 |
| 1980 | 28 | 13.8 |
| 1981 | 80 | 39.4 |
| 1982 | 44 | 21.7 |
| 1983 | 24 | 11.8 |
| Total | 203 | |

* Does not include 81 currently active projects

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partments and other groups to handle these evaluations without our assistance.

Most of our evaluations have involved government and private offices (over 75% (see Table II)), educational institutions (14.8%), and health care facilities (9.3%). Given our mandate to evaluate occupational health problems, it is not surprising that NIOSH has not investigated very many residential IAQ problems. Thus, we do not have much experience in evaluating problems which are principally encountered in residential buildings such as exposures to radon daughters or to combustion products.

In reviewing the reports on these evaluations, we have attempted to classify our findings by the type of problem found (Table III). It is of note that nearly half of these investigations have attributed the IAQ problems to inadequate ventilation. Some form of environmental contamination was thought to be the source of the problem in approximately 30%. The source of this contamination was thought to be from inside the building in 17.7% of the investigations, outside the building in 10.3%, and from the building structure in 3.4%. Problems such as hypersensitivity pneumonitis, cigarette smoking, humidity, etc., have accounted for approximately 10% of our evaluations. Finally, in another 10%, the etiology of the IAQ problem has remained unexplained.

In reviewing these results, several factors should be considered. First, over time, NIOSH has not used a standard protocol for conducting these evaluations. Our methods and criteria have changed as we became more familiar with the problem and developed new approaches. Also, some of these investigations were conducted several years ago, leaving only scanty data and a brief report for current review. In the early studies, many of the "unknown" problems may have actually been due to inadequate ventilation, but the reports did not provide enough information to determine this. Thus, there may be considerable misclassification in this list.

TABLE II
Completed NIOSH Indoor Quality Investigations by Building Type (through December 1983)

| Type | Number | % |
|---|---|---|
| Government and business officer | 154 | 75.9 |
| Schools and colleges | 30 | 14.8 |
| Health care facilities | 19 | 9.3 |
| Total: | 203 | |

TABLE III
Completed NIOSH Indoor Air Quality Investigations by Type of Problem (through December 1983)

| Problem | Number | Total |
|---|---|---|
| Contamination (inside) | 36 | 17.7 |
| Contamination (outside) | 21 | 10.3 |
| Contamination (building fabric) | 7 | 3.4 |
| Inadequate ventilation | 98 | 48.3 |
| Hypersensitivity pneumonitis | 6 | 3.0 |
| Cigarette smoking | 4 | 2.0 |
| Humidity | 9 | 4.4 |
| Noise/illumination | 2 | 1.0 |
| Scabies | 1 | 0.5 |
| Unknown | 19 | 9.4 |
| Total: | 203 | |

This listing is also not necessarily representative of the general distribution of indoor air quality problems in offices. Often, NIOSH is requested to conduct an evaluation only after initial attempts to identify the problem have failed, or complaints have persisted after initial corrections have been made. Large public-sector agencies use NIOSH as a resource, but managers of smaller offices or office buildings may be unaware of our program. Therefore, these facilities may be underrepresented on our list. Despite these shortcomings, the list does provide an overview of the types of indoor air quality problems encountered in office environments. A brief review of the major types of IAQ problems follows.

### *Contamination from inside the office environment*

This classification (approximately 18%) refers to exposure to a chemical or other toxic agent generated within the office space. Usually the symptoms experienced by the office worker are directly linked to the exposure, but if the exposure is disseminated through the building's ventilation system, localization of the source may be difficult. Examples from Health Hazard Evaluations include exposures to methyl alcohol from spirit duplicators,[1] exposures to methacrylate from copiers,[2]

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and exposures to sulfur dioxide from a heating system.[3] Occasionally, a very specific health effect can aid in the investigation, such as the occurrence of dermatitis from amines used in humidification systems.[4,5] The use of pest control agents, such as chlordane, may also cause persistent problems in office environments.[6,7]

These problems can usually be identified by inspecting the affected office area (e.g., copying machines) and by questioning about other uses of chemicals in the building (e.g., pesticides, humidification agents). Environmental and medical testing appropriate for that chemical can then be conducted.

*Contamination from outside the building*

This classification (approximately 10%) refers to exposure to a chemical or other toxic substance originating from a source outside the building. Common examples include motor vehicle exhaust either from a parking garage or loading dock entering the building through the intake from the ventilation system.[8,9] Many of these exposures are "presumed" explanations that cannot be documented at the time of the investigation. Although the dispersal of substances through the ventilation system may seem obvious, the complexity of the system may require the use of more sophisticated techniques such as a tracer gas to document the problem.[8] Other outside sources include nearby construction activity which may generate sufficient exhaust fumes, dust, or other contaminants to cause complaints in nearby offices.[10]

A few years ago, NIOSH evaluated a dramatic case of outside contamination of an office building when we discovered a 6-million-gallon underground gasoline spill while investigating irritative symptoms among the office occupants.[11] While this may be viewed as unusual, there is concern about the increasing number of underground gasoline tank leaks.

*Contamination from the building fabric*

Contamination from the building fabric (approximately 3% of our evaluations) refers to problems from the material used to construct the building. While not included in our list of evaluations, asbestos is obviously a major source of concern about indoor air quality. Other insulating materials are a common source of these problems because of the release of substances such as formaldehyde.[12] Dermatitis due to fibrous glass has also been a common problem, usually after the fibrous glass insulation has been disturbed during some construction activity.[13]

*Hypersensitivity pneumonitis*

This group (approximately 3% of our evaluations) refers to problems due to a hypersensitivity reaction to microorganisms in the building environment. Although not a common cause of office problems, the potential medical severity of this condition and the difficulties in controlling this problem make it an important cause of office problems. NIOSH's evaluations regarding this problem will be discussed later in this volume.

*Inadequate ventilation*

By far the largest classification, this group makes up approximately one-half of our completed evaluations. Our determination of inadequate ventilation is commonly made after considering a number of factors, including the absence of other sources of contamination and the presence of only very low levels of contaminants in our environmental sampling results. These, linked to the widespread occurrence of non-specific symptoms such as headaches, eye irritation, and upper respiratory irritation, suggest that an evaluation of the ventilation system may be warranted.

The evaluation of ventilation systems will be discussed later. Our methods range from obtaining specifications on the building ventilation system to detailed air flow measurements. Both approaches can present difficulties. Information on the specifications of the ventilation system is often not readily available from the building operators. Buildings with renovated ventilation system are often very difficult to evaluate because of the piecemeal approach used in such renovation. The ASHRAE guidelines for ventilation are usually used for comparison.[14] While one can question the basis for these guidelines, we have found them useful in evaluating IAQ problems and for recommending corrections where there is a problem because of inadequate ventilation.

The pathogenesis of complaints or symptoms caused by inadequate ventilation is not clear, but certain extreme situations have provided us with some insight into the relationship between such complaints and inadequate ventilation. In 1982,

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we conducted an evaluation at a government office building in Idaho with widespread complaints among the employees. Despite environmental surveys showing no significant comtaminant levels, the employees were moved to another building. Our investigators found that the air intake for the building had been covered with plastic one year earlier, to protect the air handling system from airborne debris from Mount St. Helens, and that this cover had never been removed. Removal restored intake of adequate outdoor air to the building and allowed reoccupancy without significant problems.[15] However, in this situation, no environmental measurement indicated that there was a problem. In general, we have not found any environmental measurement to be useful as an indicator of poor ventilation. However, it should be noted that other investigators have found carbon dioxide levels useful for such evaluations.[16,17] Low levels of multiple contaminants are often present in these situations and are currently the best explanation for the occurrence of symptoms. The pattern of contaminants probably varies from building to building, but we do not yet have adequate measurement techniques or adequate knowledge to easily recognize this problem through environmental sampling.

## Current evaluation methods

Our current approach to evaluating office environment requests usually begins with a walk-through evaluation by an industrial hygienist. Prior to this, we try to obtain background information on the history of the building design or construction and try to ensure that the building engineer will be present during the investigation. During the initial visit, we obtain a history of complaints among office occupants by interviewing as many as is feasible. This is helpful not only for identifying the type of medical complaints, but also for obtaining a chronology of the problem and ascertaining the time pattern of symptoms (afternoon more than morning, etc.).

Potential sources of contamination are identified during the initial walk-through evaluation. Some will be obvious (e.g., copying machines), while others may be identified only after careful questioning (e.g., pesticide spraying). We also usually inspect the ventilation system for the particular office area and attempt to understand its connection to the system for the entire building. Information on the control of the ventilation system (outdoor air intake relative to temperature, etc.) is also obtained. Some environmental sampling may be conducted if a source of contamination is found or suspected. Some general air monitoring (e.g., organics) may also be conducted, but this is usually more helpful to reassure the occupants that the toxic substances of concern to them are not present in any degree than it is for identification of a problem.

The problem may be resolved during this initial visit, but, in some instances, more extensive environmental sampling, a medical study, a ventilation assessment, or some combination of these may be required. These may be necessary either to better identify the source and extent of the problem or to alleviate the concerns of the affected employees. Once our investigations are complete, our findings and recommendations are communicated to the involved parties.

## Future activities in indoor air quality

More research into office ventilation and its effect on background levels of contaminants is necessary to provide better guidelines for evaluating and controlling indoor air quality problems. Because of the nature of these exposures, a variety of governmental agencies and private groups are involved in this research effort. Recently, the Environmental Protection Agency, the Consumer Product Safety Commission, the Department of Energy, the Department of Health and Human Services, the Tennessee Valley Authority, and several other Federal agencies formed a coordinating committee on indoor air quality research. This committee will help to coordinate Federal government indoor air quality research. Already, an inventory of IAQ-related research in the Federal government has been prepared. These agencies are also coordinating the planning of a possible large national survey of indoor air pollution and related health problems.

While much of this effort and related research may seem remote to investigating specific problems in office buildings, they may provide the basis for better guidelines for evaluating indoor air quality and for necessary corrective steps. At the same time, improved methods of assessment are needed to evaluate specific types of problems.

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NIOSH is currently developing better methods for assessing indoor air quality (e.g., microorganism levels, ventilation parameters, etc.). Other groups are working on methods for other types of assessments. Meanwhile, we will continue to evaluate indoor air quality problems and, hopefully, continue to improve our efforts in these assessments.

## References


1. Centers for Disease Control: Methyl Alcohol Toxicity in Teacher Aides Using Spirit Duplicators — Washington. *Morbidity & Mortality Weekly Report* 29:437-438 (1980).
2. National Institute for Occupational Safety and Health: *Congressman Cavanaugh's Office*. Health Hazard Evaluation Report No. HETA 80-067-754. NIOSH, Cincinnati, OH (1980).
3. National Institute for Occupational Safety and Health: *Wappingers Falls School*. Health Hazard Evaluation Report No. HETA 83-172-1409. NIOSH, Cincinnati, OH (1984).
4. National Institute for Occupational Safety and Health: *Boehringer-Ingelheim, Ltd*. Health Hazard Evaluation Report No. HETA 81-247-958. NIOSH, Cincinnati, OH (1981).
5. National Institute for Occupational Safety and Health: *Cornell University*. Health Hazard Evaluation Report No. HETA 83-020-1351. NIOSH, Cincinnati, OH (1983).
6. National Institute for Occupational Safety and Health: *Georgetown University*. Health Hazard Evaluation Report No. HETA 83-444. NIOSH, Cincinnati, OH (under study).
7. National Institute for Occupational Safety and Health: *Planned Parenthood*. Health Hazard Evaluation Report No. HETA 83-258. NIOSH, Cincinnati, OH (under study).
8. National Institute for Occupational Safety and Health: *Department of Justice*. Health Hazard Evaluation Report No. HETA 80-024-887. NIOSH, Cincinnati, OH (1981).
9. National Institute for Occupational Safety and Health: *Cincinnati Technical College*. Health Hazard Evaluation Report No. HETA 82-269-1341. NIOSH, Cincinnati, OH (1983).
10. National Institute for Occupational Safety and Health: *McCalls Publishing Company*. Health Hazard Evaluation Report No. HETA 81-097-1021. NIOSH, Cincinnati, OH (1981).
11. Centers for Disease Control: Employee Illness from Underground Gas and Oil Contamination — Idaho. *Morbidity & Mortality Weekly Report* 31:451-453 (1982).
12. National Institute for Occupational Safety and Health: *Tri-Valley Federal Credit Union*. Health Hazard Evaluation Report No. HETA 81-108-883. NIOSH, Cincinnati, OH (1981).
13. National Institute for Occupational Safety and Health: *Ellis Hospital*. Health Hazard Evaluation Report No. TA 80-080. NIOSH, Cincinnati, OH (1980).
14. American Society of Heating, Refrigerating and Air-Conditioning Engineers, Inc.: *ASHRAE Standard 62-1981, Ventilation for Acceptable Indoor Air Quality*. ASHRAE, Atlanta, GA (1981).
15. National Institute for Occupational Safety and Health: *U.S. Forest Service*. Health Hazard Evaluation Report No. HETA 81-150-994. NIOSH, Cincinnati, OH (1981).
16. Rajhans, G.S.: Indoor Air Quality and $CO_2$ Levels. *Occupational Health in Ontario*, pp. 160-167. P.L. Pelmear, Ed. Ontario Ministry of Labour (1983).
17. Nicks, J.B.: Tight Building Syndrome: When Work Makes You Sick. *Occup. Safety & Health*, pp. 51-57 (1984).


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# INDOOR AIR QUALITY

## Selected References

U.S. Department of Health and Human Services
Public Health Service
Centers for Disease Control
National Institute for Occupational Safety and Health
Division of Standards Development and Technology Transfer
4676 Columbia Parkway
Cincinnati, Ohio 45226

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## INTRODUCTION

The National Institute for Occupational Safety and Health (NIOSH) has compiled this document in response to an increasing number of requests for information about indoor air quality (IAQ), including "sick building syndrome." Included in this publication are:

1. NIOSH Congressional testimony that describes the NIOSH IAQ investigations program and summarizes the results of NIOSH research and findings on IAQ problems;

2. NIOSH guidance for conducting indoor air quality investigations;

3. NIOSH journal article on evaluating building ventilation systems; and

4. List of non-NIOSH publications on indoor air quality.

As the Federal agency responsible for conducting research and making recommendations for occupational safety and health standards, NIOSH limits its IAQ activities to the occupational environment. The U.S. Environmental Protection Agency (EPA) also conducts an IAQ program and can be contacted for information regarding both occupational and non-occupational settings. Several relevant EPA publications are included in Item 4 above and can be obtained from the EPA Public Information Center, 401 M Street S.W., Mail Code PM 211B, Washington, DC 20460, telephone 202-382-2080.

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# CONGRESSIONAL TESTIMONY

TESTIMONY OF

J. DONALD MILLAR, M.D.

DIRECTOR

NATIONAL INSTITUTE FOR OCCUPATIONAL SAFETY AND HEALTH

CENTERS FOR DISEASE CONTROL

Before the

SUBCOMMITTEE ON SUPERFUND, OCEAN AND WATER PROTECTION

COMMITTEE ON ENVIRONMENT AND PUBLIC WORKS

UNITED STATES SENATE

May 26, 1989

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I am Dr. J. Donald Millar, Director of the National Institute for Occupational Safety and Health (NIOSH) of the Centers for Disease Control, in the Public Health Service of the Department of Health and Human Services. I appreciate this opportunity to report to you on the activities of NIOSH in the area of indoor air quality.

Our experience with evaluating and understanding indoor air quality problems is based on the research and technical assistance that we have conducted under the mandates of the Occupational Safety and Health Act (OSH Act). Our knowledge, therefore, relates to the health effects on workers in nonresidential and nonindustrial workplaces, including Federal buildings, schools, and other public buildings, commercial buildings and portions of commercial vehicles occupied by workers. In addition to dealing with air quality, this knowledge base includes data on ergonomic and psychosocial problems that also affect workers.

More specifically, most of our knowledge on indoor air quality problems has been generated on the "technical assistance side" of our responsibilities under the OSH Act. This assistance is conducted under Section 20(a)(6) of the OSH Act through our Health Hazard Evaluation (HHE) Program, where we respond to requests from employers, employees, employee representatives, State and local agencies and other Federal agencies. Presented below is a brief narrative of how the HHE Program is utilized for indoor air quality investigations, and what we have learned from these investigations in regard to the extent of the problem. Additional details are shown in Enclosure I.

In the 1970's, following the Arab oil embargo, energy conservation programs were encouraged throughout the United States. The operations of buildings changed in an effort to conserve fossil fuels and operating costs. Ventilation rates were reduced and buildings were sealed to prevent infiltration of untempered outside air (hot, humid air in the summer months and cold, dry air in the winter months). At the same time, there was a revolution occurring in buildings throughout the country. Computers forced a change in office procedures and productivity leading to ergonomic and organizational stress problems. Some of these new office technologies brought with them chemical and physical hazards.

We saw the effect of the conservation measures, as well as problems associated with the shifts in office automation (e.g. video display terminals, vision problems), and concern about asbestos and radiation, through increases in requests for assistance in the HHE Program. Of the 1,200 Health Hazard Evaluations between 1971 and 1978, NIOSH investigated six indoor air quality problems or 0.5% of the total. From 1978 to 1980, the percentage of HHE's have averaged 12% of all health hazard evaluations. More recently, on an annual basis, this has increased to approximately 20%. For example, in FY 1988 through the present, NIOSH's toll free information number has received an average of approximately 60 inquiries and requests for assistance per month on indoor air quality problems. In 1988, in addition to providing background information and a copy of NIOSH's indoor air quality guidance document (Enclosure I) to most of these callers, NIOSH researchers conducted 45 field investigations.

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Table 1 presents the number of HHE investigations by building type since the Program was initiated in 1971. (These totals do not include complaints arising from asbestos contamination--the number of which also is significant--or complaints regarding radon.)

TABLE 1

NIOSH Indoor Air Quality
Investigations by Building Type
(through December 1988)

| Building Type | Number Completed | Percent of Total |
|---|---|---|
| Government and Business Offices | 426 | 80 |
| Schools and Colleges | 68 | 13 |
| Health Care Facilities | 35 | 7 |
| Total: | 529 | 100 |

We have not seen a decrease in indoor air problems and we are concerned that as the U.S. moves more and more to a service and information economy, with increases in office workers, the problems will increase.

Indoor air quality problems may arise from a variety of sources including human metabolic activity, smoking, structural components of the building and contents, biological contamination, office and mechanical equipment, and outside air pollutants that enter the building. Commonly, the symptoms and health complaints reported by workers are diverse and not specific enough to readily identify the causative agent (Table 2). The workplace environment is implicated by the fact that these symptoms can be severe enough to result in missed work, reassignment, and even termination. This causes increased anxiety among the workers and, often times, makes the investigation of these problems even more difficult.

TABLE 2

Common Health Complaints

Eye Irritation
Dry Throat
Headache
Fatigue
Sinus Congestion
Skin Irritation
Shortness of Breath
Cough
Dizziness
Nausea
Sneezing
Nose Irritation

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Although some of these episodes may be made up of several factors combined, we have classified the results found in our HHE Program by primary type of problem found: contamination from the building materials (4%); microbial contamination (5%); other contamination from inside the building (15%); contamination from outside the building (10%); inadequate ventilation (53%); and unknown (13%).

As mentioned above, in over half of our indoor air quality investigations, inadequate ventilation was found to be the cause of complaints. Heating, ventilating, and air conditioning (HVAC) conditions that can cause indoor air quality problems include migration of odors or chemical hazards between building areas, re-entrainment of building exhaust through heat recover devices or improper placement of exhaust and intake stacks, buildup of microorganisms in HVAC system components and poor odor and environmental control due to insufficient "fresh" outdoor air. The insufficient use of "fresh" outdoor air has been compounded by reduction in ventilation airflows because of energy conservation. The inadequacy of building ventilation can be evaluated by monitoring ambient carbon dioxide ($CO_2$) concentrations, temperature, humidity, and airflow. However other chemical agents from sources other than human occupants also increase with adequate building ventilation.

In 20-25% of our indoor air quality surveys, sources inside the building have been identified as the major generators of indoor air pollution. To date, common sources that have been identified include: duplicators-methyl alcohol; signature machines - butyl methacrylate; blueprint copiers--ammonia; acetic acid; pesticides; boiler additives--diethyl ethanolamine; cleaning agents; tobacco smoke and combustion gases; foam insulation, particle board, plywood, construction glues and adhesives--formaldehyde, and organic solvents; lined ventilation ducts--fibrous glass; silicone caulking and curing agents. in these situations, we have found that low concentrations of agents need to be monitored since odor thresholds, comfort and unusual stimuli may be the significant factors rather than higher concentrations where health effects have been established. Also, in many instances no evaluation criteria exist and the investigators must compare areas where complaints are frequent with areas which have no complaints to search for chemical, biological, physical, and organizational factors which may be the cause. Given the problems with identifying emission sources and the need to measure at low levels, sensitive and specific direct-reading instruments need to be followed with highly specific, low level chemical analysis in the laboratory.

To complicate the investigations concerning poor air quality, ergonomics and psychosocial issues often are encountered. For example, our research teams, composed of behavioral scientists, physicians and industrial hygienists, have investigated a series of mass illness outbreaks in various work settings for which there was no apparent physical or chemical cause. The reported symptoms are typically vague and nonspecific, and frequently are described by workers as ill defined contaminants in the workplace (e.g., bad odors, stuffy or heavy air). Questionnaire surveys and interviews of both affected and unaffected workers suggest that the expression of the symptoms may have been exacerbated by a variety of ergonomic, organizational and psychosocial stresses which increase worker job and life-dissatisfaction.

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Aside from NIOSH experiences discussed above, there have been considerable activities by other governmental and nongovernmental groups worldwide of which I am sure you are aware. Some major examples of these are as follows;

1. The National Research Council's Assembly of Life Science published "Indoor Air Pollutants" in 1981. The report lists a number of chemicals implicated in indoor air pollutant problems without assessing their importance. The Assembly also recommended that monitoring protocols and special instruments be developed to asses indoor air pollutants; that complaints of malaise, headache, stuffiness, and eye and throat irritation be studied; that the lowering of work productivity due to indoor pollution and associated discomfort be investigated; and that the influences of building design on the concentration of pollutants in commercial facilities be conducted.

2. The Environmental Protection Agency (EPA) published "EPA Indoor Air Quality Implementation Plan" in 1987 which not only described EPA's research agenda at that time, but dealt with those indoor air quality issues that concerned other Federal agencies. The document included a bibliography of indoor air quality literature containing over 2,000 entries.

3. In 1988, the Health and Safety Executive of Great Britain issued a report "Sick Building Syndrome: A Review." This report summarizes their experiences with "sick building syndrome," and discusses symptoms, common features of "sick buildings" and possible causes. Their experiences mirror that information found by our HHE Program.

4. A 1988 EPA publication, "Indoor Air Quality in Public Buildings" reported that concentrations of volatile organic compounds in new buildings were found to be as much as 100 times higher than those found outdoors.

5. Volume II of the Environmental Protection Agency's Report to Congress prepared in 1988/89 under Title IV of the Superfund Amendments and Reauthorization Act of 1986 highlights an up-to-date summary of the "Assessment and Control of Indoor Air Pollution." This volume discusses such issues as (a) factors affecting indoor air quality; (b) sources of pollutants and health effects; and (c) economic impacts.

These examples support the point that indoor air quality problems associated with worker health are significant and require continued vigilance.

In regard to your questions pertaining specifically to the Indoor Air Quality Act (S.657), the Administration has not yet taken a position on the specific contents of the proposed legislation.

Thank you for the opportunity to submit this testimony to the record.

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ENCLOSURE I

# THE NIOSH APPROACH TO CONDUCTING INDOOR AIR QUALITY INVESTIGATIONS IN OFFICE BUILDINGS

National Institute for Occupation Safety and Health

## ABSTRACT

Since 1971, personnel from the National Institute for Occupation Safety and Health (NIOSH) have complete over 500 indoor air quality (IAQ) investigations in a variety of office building environments. Most of these investigations have been conducted since 1979, paralleling the energy conservation concerns of building owners and operators. These investigations have been conducted under the authority of the NIOSH Health Hazard Evaluation Program and have been in direct response to reported health complaints or illness. Therefore, these IAQ investigations are intended to establish the identity of a problem and to recommend solutions for its correction. Over time, we have developed a consistent methodology with a "solution-oriented" approach to conducting these IAQ investigations. To initiate the investigation, the NIOSH team gathers background information by telephone and then makes a site visit to interview the affected employees and establish symptom identity and prevalence. During this initial site visit, the investigators also attempt to identify sources of contaminants, evaluate comfort parameters, and assess ventilation system performance. A variety of applicable evaluation criteria may be used, including "rules-of thumb" gleaned from the current scientific literature and our own experiences. If specific problems cannot be identified through these initial means, follow-up visits are then used to pursue a continually narrowing range of possibilities. This "solution-oriented" approach has resulted in the best allocation of our resources and has allowed the most efficient use of in-field as well as analytical personnel. In the IAQ investigations completed to date, problems were found to result from building material contaminants in 4%, microbiological contaminants in 5%, contaminants brought in from outside the building in 10%, contaminants from inside the building in 15% and inadequate ventilation problems in 53%. The remaining 13% represent those investigations where no problem could be identified.

## INTRODUCTION

The sometimes questionable quality of indoor air and the potential for health risks have become major concerns of building occupants, especially office workers. Some potential indoor exposures, such as to the carcinogen, asbestos, have well-documented health implications. But, more commonly, the health risks of other indoor air exposures are poorly understood. Nevertheless, office workers experiencing indoor air quality (IAQ) problems often demand a complete evaluation of their work environment and of the effect it may have upon their health.

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At the National Institute for Occupation Safety and Health (NIOSH), the majority of our indoor air quality investigations are conducted as part of the Health Hazard Evaluation Program. We conduct these health hazard evaluations at the request of employee groups, unions, management, and local, State and Federal agencies. Generally, these requests are in response to existing worker health complaints and illness. Because we are essentially "invited" to conduct these investigations, the data presented here will not reflect a statistically valid cross-section of the indoor air quality problem. However, these findings are drawn from one of the largest single information bases currently available on the subject. In essence, this paper will summarize the NIOSH methodology currently used during IAQ investigations and some of the data from these IAQ investigations completed since the start of the Health Hazard Evaluation Program.

METHODOLOGY

We have found that investigating IAQ problems can present a formidable challenge which, in some ways, is more difficult than evaluating industrial environments. In an industrial situation, the evaluation will be directed by investigations of the materials used by, or in the vicinity of, the affected workers. These materials can usually be chemically analyzed which permits techniques and potential health effects using standard medical and epidemiologic techniques. Frequently there are exposure criteria which can be applied to help interpret the data obtained. This is rarely the case with an indoor air quality problem.

Indoor air quality investigations tend to become more complicated as time passes without identification of a cause. Frustrations result in highly charged emotions which only further impede continued evaluation efforts. These situations are further complicated by the fact that symptoms are not easily attributed to a single cause and the application of standard industrial hygiene, medical and epidemiologic techniques may prove to be inconclusive.

Over time, our approach in evaluating this situation has changed. We have developed a more consistent, solution-oriented approach that systematically excludes a continually narrowing range of possibilities. Generally, this exclusion hierarchy, which has come about based on our past experiences, involves evaluation of physical, chemical and microbiological factors, in the order presented. Each of these potential causation categories are discussed in more detail later in this presentation.

Since we anticipate that IAQ requests will continue to represent a substantial percentage of the total health hazard evaluation requests (currently about 20%), three response levels have been developed. Based on the information obtained during initial telephone with the requestor, the following responses are possible:

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1. Provide "self-help evaluation" materials (attached). Remain available for consultation by telephone. Become more involved, if necessary.

2. NIOSH conducts an initial evaluation an provides recommendations to solve the problem or for further study on a "self-help" basis.

3. NIOSH conducts a full scale investigation.

The NIOSH investigation team commonly includes an industrial hygienist and physician/epidemiologist, but can also include other professionals such as an engineer. Most investigations contain the following parts: background assessment, initial site assessment, and, if necessary, additional site assessment.

BACKGROUND ASSESSMENT

For the background assessment, we initially obtain, by telephone, as much information on the building as practicable, an idea of symptoms being experienced, and a chronology of the problem. Much of this information can be collected using a standard questionnaire. We also request copies of other previous investigations which are relevant to the problem at hand. These data are then used to tailor the protocol for the initial site assessment so as to make it more efficient.

INITIAL SITE ASSESSMENT

For the initial site assessment, a common protocol includes five separate steps or parts: an opening conference, a walk-through evaluation, personal interviews, phase I of environmental monitoring, and a closing conference.

Opening Conference--The opening conference is attended by representatives of the employer and employees (where applicable) as well as someone who has knowledge of the operation, and maintenance of the building's heating, ventilating and air conditioning (HVAC) systems. This meeting serves to present NIOSH's role, discuss anticipated activities and arrange to receive copies of pertinent data not already received.

Walk-through Survey--The walk-through survey will involve all or part of the building including inspection of the HVAC systems with special attention given to the mechanism by which outside air enters each HVAC unit. Architectural plans and ventilation test and balance reports may also be reviewed during this phase. Potential sources of emission are identified so that each may be further evaluated, as needed.

Personal Interview--Personal interviews are often conducted to determine the extent, prevalence and character of reported symptoms. The use of a questionnaire, such as the one shown in Appendix 1, may be the most efficient means of collecting this type of information. It can be used as a guide during personal interviews or it can be self-administered

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Phase I Environmental Monitoring--Phase I environmental monitoring is usually conducted on each initial survey. The scope of this effort may vary, but usually will either evaluate certain aspects of the building environment, which we have come to believe are important factors in all investigations, or explore any other possibilities made apparent from the background assessment. Evaluation of the ventilation system using both actual measurement and/or carbon dioxide (CO2) techniques, and monitoring temperature and relative humidity are useful procedures for all evaluations during the initial site assessment. Monitoring for formaldehyde is an example of a specific method which may be used if the background assessment indicates that respiratory system and eye irritation are prevalent complaints and the space has been recently built or renovated (a number of furnishings are potential sources of formaldehyde). Most of the monitoring accomplished on the initial survey is obtained using direct-reading instruments where possible because they provide results on-the-spot. Any deficiencies noted can be corrected and re-evaluated. Trace concentrations of hundreds of compounds could be identified depending on the extent of the sampling and analytical effort; however, the concentrations usually detected would not be expected to cause adverse health effects in a normal healthy individual. Other techniques which I will now discuss have been consistently more useful.

Evaluating HVAC Systems--HVAC systems can be complicated and most industrial hygienists have received very little or no training in the design, maintenance and trouble-shooting of building ventilation systems. The most important aspect of evaluating HVAC systems is to gain an understanding of how they are supposed to be working and then use some relatively simple methods to convince yourself that the system is performing up to the design specifications, and whether this is adequate with respect to the complaint areas in the building, return to the complaint area(s) and measure supply and return air flows using either a velometer or a flow hood and compare the results to the design quantities. Note that in variable-air-volume (VAV) systems the supply air flows may vary during the day.

A method which is gaining popularity, and which is currently used by NIOSH for evaluating the adequacy of ventilation to an area is the measurement of CO2 concentrations. Humans expire significant quantities of CO2. The higher the CO2 levels inside a building, the poorer the overall ventilation, in a general sense. We believe that complaints will not be prevalent if interior CO2 concentrations are maintained at twice or less the outdoor levels (usually 250-300 ppm). At CO2 concentrations above 1000 ppm, or 3 to 4 times the outdoor level, complaints of headaches tiredness, eye, nose and throat irritation may be more prevalent. It is important to realize that it is not the CO2 concentration that is causing the symptoms; but, if CO2 increase, so may all the other normal airborne contaminants and it may be some combination of all these substances that make people uncomfortable. Carbon dioxide measurement scan be obtained using standard detector tubes or portable CO2 monitoring instruments. The use of CO2 as an index of the general quality of indoor air is currently being evaluated by NIOSH as well as many other agencies.

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Poor mixing of air is another potential problem sometimes found when air is delivered and returned through ceiling diffusers. Standard smoke tubes, and temperature and CO2 measurements at 1 foot and 7 foot heights above the floor may be useful techniques in evaluating stratification resulting from poor mixing.

Monitoring Temperature and Relative Humidity--Temperature too cold, too warm or fluctuating can be a source of complaints. While individuals vary in their limits of thermal comfortability, if a significant number of workers in an area complain (more than 20%), then temperature and relative humidity may be creating an uncomfortable environment. If temperatures are too warm, complaints or tiredness, lack of concentration and headache may also be reported. Low relative humidities, not uncommon in the winter in a building or residence that is not humidified, can cause eye, nose and throat irritation.

## CLOSING CONFERENCE

The closing conference of the initial site survey provides an opportunity to present the NIOSH activities accomplished, any results obtained and recommendations on corrective actions if potential problems were identified. If no problems were identified, recommendations may be made on how to continue studying the problem either on a self-help basis or through continued NIOSH involvement. Typical recommendations when we have not identified a probable explanation for the reported symptoms would include the formulation of a more formalized method of reporting worker symptoms on a daily basis and the generation of HVAC data logs to provide evidence that the HVAC system is performing in a consistent manner over time.

## EVALUATION CRITERIA

Evaluation criteria used to interpret environmental measurements vary. In the classic industrial hygiene sense, the Occupational Safety and Health Administration's (OSHA) permissible limits [1], the American Conference of Governmental Industrial Hygienists' (ACGIH) Threshold Limit Values [2], and NIOSH's recommended exposure limits [3] are most commonly used in occupational exposure assessments. Because these criteria are based on health effects as they pertain to the manufacturing environment, they may not have the same relevance for workers in an office setting, whose primary concern may be for comfort or simply an absence of unusual sensory stimuli over their working period. The Environmental Protection Agency (EPA) has ambient air quality standards [4] for a variety of pollutants designed to protect the public over an entire day (not just an 8-hour workday). However, these, too, may not have relevance to an indoor office environment, especially from the perspective of problem-solving.

The American Society of Heating, Refrigerating and Air Conditioning Engineers, Inc. (ASHRAE) guidelines for acceptable indoor air quality [5] have been developed for the indoor environment. We commonly use these as criteria in our office building evaluations, especially for assessing the performance of a ventilation system. We also use the ASHRAE comfort guidelines [6] as criteria for assessing the thermal performance of occupied space.

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## ADDITIONAL SITE ASSESSMENTS

If the background assessment and the initial site visit identify a problem that need further definition, or if no problem can be isolated, an additional site assessment may be performed. During the additional site assessment, more specific and extensive monitoring may be conducted for chemical and/or microbiological contaminants of interest. Sampling for airborne microbiological contaminants is also outside of what is considered a standard industrial hygiene technique. Useful techniques are still under evaluation. Methodology is available; however, analytical support is somewhat limited. Nevertheless, the topic of potential microbiological contamination usually comes up as an investigation progresses. Close visual inspection of the various HVAC components will usually uncover a microbiological problem if present.

The condition most commonly associated with exposure to airborne organisms is hypersensitivity pneumonitis. This is a general term for a disease which occurs as a result of an immunologic inflammatory reaction to the inhalation of any of a variety of organic dusts. Terms like humidifier fever, ventilation pneumonitis, farmer's lung and cheese worker's lung are all the result of these exposures. Symptoms are usually described as a recurring "flu-like" syndrome. Diagnosis is based on a combination of characteristic symptoms, chest x-rays, pulmonary function abnormalities and sometimes immunologic studies [7].

Inspection of the HVAC system and confirmation of the diagnoses of hypersensitivity pneumonitis among workers may be more useful than air sampling for airborne microorganisms until investigative techniques are further refined.

## DISCUSSION

Through December 1988, 529 NIOSH indoor air quality health hazard evaluations have been completed (Table 1). These do not include our investigations of asbestos-in-building problems, but only those where the building occupants were actually experiencing ill health effects. The number of investigations has increased markedly since 1979. This is most probably due to a couple of factors: increased energy conservation measures and increased worker awareness of their office environment. We now average about two indoor air quality investigations per week.

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Source: https://www.industrydocuments.ucsf.edu/docs/zqyx0000

TABLE 1

NIOSH INDOOR AIR QUALITY INVESTIGATIONS
BY YEAR (THROUGH DECEMBER 1988)

| Year | Number Completed | % |
|---|---|---|
| Pre-1978 | 6 | 1 |
| 1978 | 9 | 2 |
| 1979 | 12 | 3 |
| 1980 | 28 | 6 |
| 1981 | 82 | 18 |
| 1982 | 52 | 12 |
| 1983 | 61 | 14 |
| 1984 | 56 | 13 |
| 1985 | 81 | 18 |
| 1986 | 59 | 13 |
| 1987 | 38 | 8 |
| 1988 | 45 | 9 |
| Total: | 529 | 100 |

While the majority of our investigations have been conducted in government and private-sector office buildings (Table 2), we have also looked at problems in schools, colleges, and health care facilities.

TABLE 2

NIOSH INDOOR AIR QUALITY INVESTIGATIONS
BY BUILDING TYPE (THROUGH DECEMBER 1988)

| Building Type | # Completed | % |
|---|---|---|
| Government and Business Offices | 426 | 80 |
| Schools and Colleges | 68 | 13 |
| Health Care Facilities | 35 | 7 |
| Total: | 529 | 100 |

Commonly, the symptoms and health complaints reported by the office workers are diverse and not specific enough to readily identify the causative agent (Table 3). The workplace environment is implicated by the fact that these symptoms normally disappear on weekends away from the office. At times, these symptoms can be severe enough to result in missed work, reassignment, and even termination. This causes increased anxiety among the workers and, often times, makes the investigation of these problems even more difficult and frustrating.

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Source: https://www.industrydocuments.ucsf.edu/docs/zqyx0000

TABLE 3

COMMON HEALTH COMPLAINTS

| | |
|---|---|
| Eye Irritation | Shortness of Breath |
| Dry Throat | Cough |
| Headache | Dizziness |
| Fatigue | Nausea |
| Sinus Congestion | Sneezing |
| Skin Irritation | Nose Irritation |

Although many of these problems may be multifactorial, we have classified our evaluations by primary type of problem found: contamination from the building material (4%); microbiological contamination (5%); contamination from outside the building (10%); contamination from inside the building (15%); inadequate ventilation (53%); and unknown (13%) (Table 4.) There are some shortcomings to these data, however, in that they may not represent a "true" cross-section of the indoor air quality problem as previously discussed. For example, we have not used a standard protocol for all these evaluations, as our methods and criteria have evolved with time and experience. Also, since many of these investigations were reviewed retrospectively, there may be some misclassification due to the vagueness of earlier reports. Lastly, we have little follow-up data on many of these evaluations to enable us to determine the efficacy of our recommendations[8].

TABLE 4

NIOSH INDOOR AIR QUALITY INVESTIGATIONS
BY PROBLEM TYPE (THROUGH DECEMBER 1988)

| Problem Type | # Completed | % |
|---|---|---|
| Building Materials Contamination | 21 | 4 |
| Microbiological Contamination | 27 | 5 |
| Outside Contamination | 53 | 10 |
| Inside Contamination | 80 | 15 |
| Ventilation Inadequate | 280 | 53 |
| Unknown | 68 | 13 |
| Total: | 529 | 100 |

BUILDING MATERIALS CONTAMINATION

Contamination from building materials and products was the major problem in 4% of our investigations. Formaldehyde can off-gas from urea-formaldehyde foam insulation, particle board, plywood, and some glues and adhesives commonly used during construction. Other building fabric contamination problems encountered included dermatitis resulting from fibrous glass, various organic solvents from glues and adhesives, and acetic acid used as a curing agent in silicone caulking.

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Source: https://www.industrydocuments.ucsf.edu/docs/zqyx0000

## MICROBIOLOGICAL CONTAMINATION

Five percent of our investigations have involved some type of microbiological contamination. Even though this is not a common cause of office problems, it can result in a potentially severe condition known as hypersensitivity pneumonitis. This respiratory problem can be caused by bacteria, fungi, protozoa, and microbial products that may originate from ventilation system components. A similar condition known as humidifier fever, most commonly reported in Europe, is also a result of microbiological contamination in ventilation systems. In our investigations, microbiological contamination has commonly resulted from water damage to carpets or furnishings, or standing water in ventilation system components.

## OUTSIDE CONTAMINATION

Contamination from sources outside the office space was the major problem identified in 10% of our investigations. Problems due to motor vehicle exhaust, boiler gases, and previously exhausted air are essentially caused by re-entrainment. This is usually the result of improperly locked exhaust and intake vents or periodic changes in wind conditions. Other outside contamination problems include contaminants from construction or renovation such as asphalt, solvents, and dusts. Also, gasoline fumes infiltrating the basement and/or sewage system can sometimes be a problem and are usually caused by gasoline leaks from ruptured underground tanks at nearby service stations. One of the most common sources of outside contamination has been carbon monoxide fumes from basement parking garages being recirculated through the building ventilation system.

## INSIDE CONTAMINATION

Contamination generated by sources inside the office space was the major problem identified in 15% of our investigations. Copying machines are often found to be a significant source. Examples of this type of problem would include methyl alcohol from spirit duplicators, butyl methacrylate from signature machine and ammonia and acetic acid from blueprint copiers. Still other inside contamination problems we have encountered include pesticides which were improperly applied; dermatitis from boiler additives such as diethyl ethanolamine; improperly diluted cleaning agents such as rug shampoo; tobacco smoke of all types; combustion gases from sources common to cafeterias and laboratories; and cross-contamination from poorly ventilated sources that leak into other air handling zones.

Contaminants from inside or outside the office space, and from the building fabric are essentially chemical contaminants. Many times odors are associated with some of these contaminants which may aid in source identification. Additionally, in most cases, these chemical contaminants were measured at levels above ambient but far below any existing occupational standard.

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Source: https://www.industrydocuments.ucsf.edu/docs/zqyx0000

## INADEQUATE VENTILATION

In 53% of our investigations, the building ventilation has been inadequate. When evaluating building ventilation, we normally use ASHRAE standards for comparison. ASHRAE standards 62-1981, "Ventilation for Acceptable Indoor Air Quality" (ASHRAE 1981) and 55-1981, "Thermal Environmental Conditions for Human Occupancy" (ASHRAE 1981) are both used. Some of the ventilation problems we commonly encounter are: not enough outdoor air supplied to the office space; poor air distribution and mixing which causes stratification, draftiness, and pressure differences between office spaces; temperature and humidity extremes of fluctuations (sometimes caused by poor air distribution); and air filtration problems caused by improper or no maintenance to the building ventilation system. In many cases, these ventilation problems are created or enhanced by certain energy conservation measures. These include reducing or eliminating outdoor air; reducing infiltration and exfiltration; lowering thermostats in winter, raising them in summer; eliminating humidification or dehumidification systems; and early shut-down and late start-up of ventilation systems.

## CONCLUSION

The major problems identified in these NIOSH indoor air quality investigations can be placed into three general categories listed with decreasing frequency: inadequate ventilation, chemical contamination, and microbiological contamination. Inadequate ventilation is the single largest problem we have seen in buildings. Although varied, these ventilation problems commonly can allow a build-up of any contaminants present in the occupied space to the point that adverse health effects are experienced or allow the environment to become annoyingly uncomfortable to the office workers. As our experience increased over time, we developed a solution-oriented approach to conducting these evaluations which places a high priority on building ventilation. This approach has resulted in the best allocation of our resources and has allowed more efficient use of in-field as well as analytical time.

Increasing office worker awareness and the current shift to office-based, service-type employment will no doubt increase concerns about the indoor air quality in offices and other non-industrial settings. More research into office building ventilation and its effect on background levels of contaminants will be necessary to provide additional and more appropriate guidelines for the evaluation and control of indoor air quality problems in the future.

Early recognition of a problem, with a timely and systematic evaluation, are key factors to a quick and effective resolution.

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Source: https://www.industrydocuments.ucsf.edu/docs/zqyx0000

REFERENCES


[1] OSHA, OSHA Safety and Health Standards, Occupational Safety and Health Administration, Washington, D.C., 1983.

[2] ACGIH, Threshold Limit Values for Chemical Substances and Physical Agents in the Workroom Environment and Biological Exposure Indices with Intended Changes for 1985-1986, Cincinnati: American Conference of Governmental Industrial Hygienists, 1985.

[3] CDC, "NIOSH Recommendations for Occupational Safety and Health Standards," Morbidity and Mortality Weekly Report, Vol. 35, 1985, pp. 35-315.

[4] EPA, National Primary and Secondary Ambient Air Quality Standards, Washington, D.C.: Environmental Protection Agency, 1971.

[5] ASHRAE, ASHRAE Standard 62-1981, "Ventilation for Acceptable Indoor Air Quality," Atlanta: American Society for Heating, Refrigerating and Air-Conditioning Engineers, Inc., 1981.

[6] ASHRAE, ANSI/ASHRAE Standard 55-1981, "Thermal Environmental Conditions for Human Occupancy," Atlanta: American Society for Heating, Refrigerating and Air-Conditioning Engineers, Inc., 1981.

[7] Levy, M.B. and Fink, J.N., "Hypersensitivity Pneumonitis," Annals of Allergy, Vol. 54, 1985, pp. 167-171.

[8] Melius, J., Wallingford, K., Keenlyside, r., Carpenter, J., "Indoor Air Quality--the NIOSH Experience," Annals of the American Conference of Governmental Industrial Hygienists, Vol. 10, 1984, pp 3-7.


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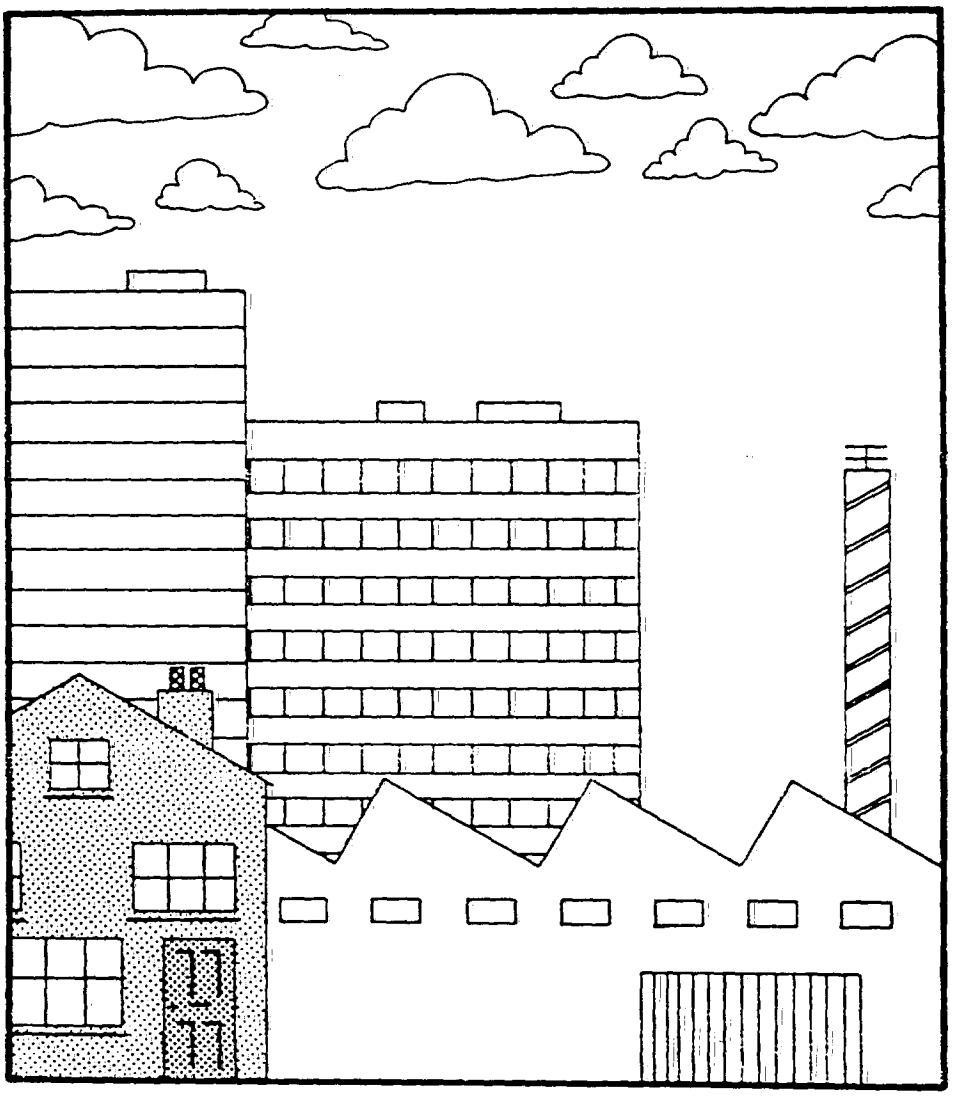
# INDOOR AND AMBIENT AIR QUALITY



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# INDOOR AND AMBIENT AIR QUALITY

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# SOURCE, NATURE AND SYMPTOMOLOGY OF INDOOR AIR POLLUTANTS

G.Robertson

ACVA Atlantic Inc, 10378 Democracy Lane, Fairfax, VA 22030, USA

## INTRODUCTION

ACVA Atlantic Inc. specializes in the study of indoor air pollution. Since we established ACVA in 1981, we have pioneered a multi-disciplined approach to the investigation of internal pollution. Investigators include chemists, microbiologists, and air conditioning engineers -- three disciplines unused to working as a team. Our client list includes numerous government agencies; multi-national companies in insurance, finance, industry, banking, and property management; colleges, schools, and numerous hospitals. Most of our clients now not only ask us to examine other buildings that they own, but also enter into long term contracts of regular monitoring and preventive maintenance. In fact, as of March 1988, we have now studied the indoor air quality of over 42 million square feet of property.

## INDOOR POLLUTANTS - THE SOURCES

Virtually everything we use in the interior sheds some particulates and/or gases. When a building is new, some compounds are given off quickly and soon disappear. Others continue "off-gassing" at a slow pace for years. Common office supplies and equipment have been found to release dangerous chemicals-especially duplicators and copiers and we have even found formaldehyde being released from bulk paper stores.

People themselves are a major contributor since each person sheds literally millions of particles, primarily skin scales, per minute. Many of these scales carry microbes but fortunately the vast bulk of these microbes are short lived and harmless.

Clothing, furnishings, draperies, carpets, etc. contribute fibers and other fragments. Cleaning processes, sweeping, vacuuming, dusting, etc. normally remove the larger particles, but often increase the airborne concentrations of the smaller particles. Cooking, broiling, grilling, gas and oil burning, smoking, coal and wood fires also generated vast numbers of airborne particulates, vapors, and gases. If the windows and doors are closed all of these can only accumulate in that internal environment.

## INDOOR POLLUTANTS -- THE TYPES

There are many types of indoor pollutants, gases, vapors, dusts, fibers, and viable and non-viable microorganisms. Some of the more common ones are described below.

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Organic Chemicals

There are arguably the widest range of pollutants with literally thousands of specific types fortunately occurring in very dilute concentrations which are usually expressed as parts per million or per billion. Most of these are presumed to be safe at the very low levels encountered, although some synergism between different organics or some incidences of organics "sensitizing" people to other pollutants cannot be ruled out. Usually the organics are more a problem in the typical home than the office and concentrations in the home are usually higher than the office mainly due to lower air exchange rates.

Radon Gas

Radon, a decay product of uranium, is present in variable quantities in soils. It moves from the soil by diffusion into the soil's air pockets or into soil water. Then the radon can migrate from the soil air through unvented crawl spaces, building foundation cracks, etc. into the indoor space. Some building aggregates, cinder block, etc. also contain radon and out-gassing from these materials add to the indoor air levels. In other cases radon enters a building via the water supply. Some of this radon is released when there is turbulence of the water such as a running tap. It has been estimated by some researchers that anywhere from 10 to 15% of the average radon we are exposed to comes from such water. However, the general consensus is that the principal source of radon in buildings undoubtedly is the soil gas. Pollution by radon is far more prevalent in homes than in offices, again mainly due to the lower air exchange rates in homes plus the fact that homes have a larger area of exposure to soil relative to building volume and soil leakage area.

Inorganic Oxides

Carbon dioxide is produced by respiration and combustion, oxides of nitrogen and sulphur are combustion products associated with gas stoves, wood, coal fires, and kerosene heaters. Carbon monoxide is emitted from unvented kerosene heaters or wood stoves and it frequently diffuses into buildings from automobile exhaust fumes generated in adjacent garages. Small to trace quantities of each of these gases and other organics are present in cigarette smoke.

Ozone is another gas that is generated, usually in very small quantities, by miscellaneous copying machines and by certain electrostatic precipitators that are used to clean up the air. In one specific case that we studied, the maintenance staff of a building switched off the main air supply fans over the weekend, but omitted to switch off the central electrostatic precipitators. Thus, ozone accumulated inside the air handlers and was subsequently delivered to the staff first thing each Monday morning. When the fans were switched on this caused a severe, though temporary, period of discomfort to the people working in the areas involved.

FIBERS

Asbestos

Prior to 1973, asbestos was the material of choice for fire-proofing, thermal insulation, and sound insulation. It was used as a spray-on insulation of ceilings and steel girders; as a thermal insulation of boilers, pipes, ducts, air conditioning units, etc.; as an abrasion resistant filler in floor tiles, vinyl sheet floor coverings, roofing, and siding shingles; as a flexible, though resistant, joining compound and filler of textured paints and gaskets; as a bulking material with the best wear characteristics for



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automobile brake shoes, and in countless domestic appliances such as toasters, broilers, dishwashers, refrigerators, ovens, clothes dryers, electric blankets, hair dryers, etc. In fact, the EPA has estimated that approximately 733,000 or 20% of all government, residential, and private non-residential buildings in the U.S. contain some type of friable asbestos-containing material.

The fact is that many asbestos bearing materials or products are of no health risk whatsoever when used in the normal course of events. However, if for any reason of wear, abrasion, friability, water damage, etc., any of the asbestos fibers are released into the air and inhaled into people's lungs, there is a health hazard. The scientific evaluation of all available human data provides no evidence for a "safe" level of airborne asbestos exposure, thus any quantity should be considered potentially dangerous.

Glass Fibers and Other Man-made Fibers

The glass fiber (usually referred to as fiberglass) industry is in its infancy compared with asbestos and since asbestos related illnesses only manifest themselves tens of years after exposure, there are some schools of thought that suggest glass fiber fragments will also accumulate in the lungs and cause later problems. This may be so, but it is unlikely to be anywhere near as severe. The fibers of glass are not shed in such large quantities as asbestos and most of the resins, etc. bonding the fibers together appear to be extremely effective and long lasting. However, some fragmentation does occur and this is especially noticeable when the loose fiberglass insulation, popularly used in attics and ceiling voids, is disturbed. Most of us have experienced itching on contact with fiberglass and dermatitis-type reactions are not infrequent due to airborne fiberglass particles.

## MICROBES

In our reviews of the literature, the one area of indoor pollution that has received least study or research has been contamination due to microbes. Nine percent of the first 223 major buildings studied by ACVA have exhibited high levels of potentially pathogenic or allergy causing bacteria, including Actinomyces and Flavobacterium species. In addition, Legionella pneumophila, the cause of the dreaded Legionnaires' disease has frequently been isolated from inside air conditioning systems.

Perhaps more significantly, we have found over twenty-eight different species of fungus contaminating air handling systems (see Table 1).

Table 1. Fungi Isolated from Air Conditioning Systems by ACVA Systems -- 1981 to 1987

| | |
|---|---|
| Alternaria sp. | Aspergillus sp. |
| Aureobasidium sp. | Candida sp. |
| Cephalosporium sp. | Chaetomium sp. |
| Chrysosporium sp. | Cladosporium sp. |
| Curvularia sp. | Diplosporium sp. |
| Fusarium sp. | Helminthosporium sp. |
| Monilia sitophila | Monosporium sp. |
| Mucor sp. | Mycelia sterila |
| Oospora sp. | Paecilomyces sp. |
| Penicillium sp. | Phoma sp. |
| Rhizopus sp. | Rhodotorula sp. |
| Saccharomyces sp. | Scopulariopsis sp. |
| Streptomyces sp. | Tricothecium sp. |
| Verticillium sp. | Yeasts |

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Of the 223 buildings studied by ACVA between 1981 and 1987, thirty-four percent have been found to contain high levels of potentially pathogenic or allergy causing fungi, including Alternaria, Aspergillus, Cladosporium, Fusarium, and Penicillium species. In many buildings with excessive staff complaints, either Aspergillus and/or Cladosporium species of fungus were found growing to excess in the air conditioning ductwork systems. In some investigations, epidemiological tests run by various doctors have confirmed severe allergic reactions to the spores of these fungi in all affected staff. Subsequent cleaning and removal of the sources of these fungal contaminants have resulted in a complete abatement of complaints.

## DIRT IN DUCTWORK

HVAC systems also have been found to be poorly designed and negligently maintained. Excessive dirt accumulations are common in ductwork, even in hospitals. Frequently dirt is built into the systems during construction since the ducts are installed long before the windows, etc. and construction dusts from the site, plus wood shavings, lunch packets, coke and beer cans, etc. find themselves brushed into the vents then "out of sight -- out of mind." Thereafter over the life of the building, more dirt enters with the supply and return air. Good filters reduce the rate of this accumulation, but the only perfect filter would be a brick wall. All filters, even the ultra-efficient HEPA filters used in hospital operating rooms allow fine particles through. Many of these fine particles coalesce, sticking to each other by adhesion or electrostatic attraction and larger particles simply grow with time. In commercial buildings, much cheaper and far less efficient filters are common. Many will stop birds and moths, but that is about all. Occasionally we find that the filters have been omitted and very frequently we find they are undersize, resulting in large air gaps that allow massive volumes of air bypass to occur. Then, there are the large electrostatic precipitators that theoretically provide ultra-efficient air. In one major building we found 16 out of their 18 precipitators were inoperative due to broken parts, many had not worked for over a year. In a major hospital, we found the power pack was missing from one of these units. When inoperative electrostatic precipitators provide zero filtration.

Dirty ductwork is a prefect breeding ground for germs. It provides an enclosed space, constant temperature, humidity, and food -- which is the dirt. No germ could wish for more!

The extent of this potential problem is huge and it is very surprising what we have found in ducts. Dead insects, molds, fungi, dead birds and rodents are common. In 1984 we found two dead snakes in air supply ducts. We have also found rotting food, builders rubble, rags, and newspapers. All of these contaminate the air we breathe. It is the dirt that encourages germs to breed -- germs which cause infections.

The dirt and dusts also may be allergenic, in fact most of the dusts are, by definition, household dusts which are notorious for causing allergies in many people.

In a survey of a 750,000 square foot hospital in Virginia, we found 14 miles of ductwork. Here are a few examples of the problems we encountered in that maze of ducts. Smoke detectors blocked by dirt and inoperative; fire dampers jammed open by dirt -- they were unable to close; reheat coils completely blocked by dirt sealing off the fresh air supply; turning vanes and even the exhaust grilles completely sealed with dirt accumulations -- in the operating suite the exhaust fan was still working against these duct blockages causing such immense negative pressure in the ducts that the ducts were boving inward almost to the point of collapse; huge excesses of bacteria and fungi were present inside the air handling chambers and throughout the ductwork; cross infection rates were high and nurses, doctors, and patients complained

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about poor air quality. We have since cleaned all the air handlers and the 14 miles of ducts and have overseen the installation of more efficient filter systems. That hospital has been dramatically improved and its air quality is now well above average.

## SYMPTOMOLOGY OF INDOOR AIR POLLUTANTS

In general, when one hears of a polluted building or a so-called "sick building," one hears familiar symptoms from occupants including eye and nose irritation, fatigue, coughing, rhinitis, nausea, headaches, sore throats, and general respiratory problems. Without doubt, the pollutant most often blamed for these symptoms by the public is environmental tobacco smoke (ETS). However, there are usually confounding variables presented by a number of potential contaminants that precludes a quick analysis establishing a single source of contamination. The main problem being the incredible similarity between symptoms from widely different irritants or even environmental conditions. For example, identical symptoms have been reported for individuals exposed to formaldehyde, ammonia, oxides of nitrogen, and ozone. In addition, similar symptoms are reported by those individuals suffering allergic type reactions to numerous dusts and to microbial spores such as Aspergillus, Penicillium, and Cladosporium fungi, among others. Similar symptoms have been reported from exposure to cotton dust and fiberglass fragments and an ever increasing and similar problem is encountered due to low relative humidities. The latter is well known to frequent flyers of airliners where relative humidity levels are frequently as low as 10%, compared to a normal lower comfort level of say 40%.

This similarity of symptoms is usually unappreciated by the public and in part it accounts for a bias against tobacco smoke, which happens to be the sole visible air pollutant. Furthermore, due to their unreliability, we, as a policy, refuse to rely upon or otherwise use the information generated by subjective building occupant questionnaires. Only upon careful investigation of the entire indoor environment and ventilation system of a building can be draw informed conclusions about the various causes of poor indoor air quality. As a result, we have made it our business to perform precisely such investigations. Despite being the main suspect of the occupants in many of the buildings we have examined, we have determined high levels of environmental tobacco smoke to be immediate cause of indoor air problems in only four percent of the 223 major buildings investigated by ACVA between 1981 and 1987 (see Table 2). Significantly, in those few cases where high accumulations of ETS have been found, ACVA also has discovered an excess of fungi and bacteria in the HVAC system. These microorganisms usually are found to be the primary causes of the complaints and acute adverse health effects reported by building occupants.

Table 2. ACVA Systems Experience -- 1981 to 1987.

| | |
|---|---|
| Total building studies | 223 |
| Number of square feet | 39,000,000 |
| Estimated number of occupants | 225,000 |

Summary of most significant pollutants found:

| Major Pollutants in Air | # of Buildings |
|---|---|
| Allergenic Fungi | 34 |
| Allergenic or pathogenic bacteria | 9 |
| Glass fiber particles | 7 |
| Tobacco smoke | 4 |
| Carbon monoxide (vehicles) | 3 |
| Miscellaneous gases | 2 |



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| Carbon monoxide (vehicles) | 3 |
| Miscellaneous gases | 2 |

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## VENTILATION AND INDOOR POLLUTION

The fact is that the accumulation of many pollutants is itself a symptom of a more serious problem -- a problem of inadequate ventilation. Medicine teaches us that treating the symptoms simply does not work, one has to go after the cause of the problem.

Improper ventilation can sometimes be carried to extremes. The fresh air dampers were closed completely in over 35% of those buildings studied by ACVA (see Table 3). Three years ago we found a building where the "maintenance engineer" had bricked up the fresh air vents to save energy. In Washington State, one NIOSH investigator of a sick building found heavy duty polyethylene

Table 3. Sick Building Syndrome Causes -- ACVA Experience

| | | |
|---|---|---|
| Sample Buildings: | | 223 |
| totalling 39,000,000 square feet | | |
| Period: | | 1981-1987 |
| (1) | Poor Ventilation | |
| | No fresh air | 35% |
| | Inadequate fresh air | 64% |
| | Poor distribution of air | 46% |
| (2) | Poor Filtration | |
| | Low filter efficiency | 57% |
| | Poor design | 44% |
| | Poor installation | 13% |
| (3) | Contaminated Systems | |
| | Excessively dirty ductwork | 38% |
| | Condensate trays | 63% |
| | Humidifiers | 16% |

sheets sealing off the fresh air intakes. It turned out that these had been installed two years earlier to reduce the levels of silica dust being carried into the building from Mount St. Helens. There are also numerous incidences of inadequate ventilation due to hidden blockages inside ducts. Using fiber-optic technology, we have found many classical examples of such where turning vanes, dampers, and reheat coils inside ducts have been totally sealed with massive accumulations of dirt, loose insulation, etc.

Perhaps the most serious problem of ventilation is that there is no effective legislation mandating the uniform use of minimum fresh air requirements. Certainly some authorities do specify ventilation rates at the design stage -- most of these are based on ASHRAE or BOCA standards. However, the major problem is that there is no legislative structure, nor is there a practical policing methodology to ensure that the operators of buildings run their ventilation systems according to such designs.

## THE EFFECT OF ENERGY CONSERVATION

Some of these examples of inadequate ventilation were due to ignorance or accidents, however, the complex of symptoms that I have mentioned -- the "sick building syndrome" -- may result primarily from energy conservation efforts to seal buildings and reduce the infiltration/exfiltration of air. Such efforts have reduced the natural infiltration of fresh air that previously existed in many buildings, exacerbating the often undiscovered problem of a poorly designed or maintained HVAC system.



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In addition to tightening buildings and sealing windows, building managers have shut down air conditioning systems at night and on weekends in an effort to lower energy costs. When the air conditioning is shut down in humid climates, condensation builds up and settles inside the ductwork. If dirt is present in damp ductwork, spores and microbes can flourish, only to be spread throughout the building once the HVAC system is turned on the next morning. This often results in Monday morning complaints of building odors or building sickness that disappear during the week, only to recur the following Monday morning. To save more energy, automatic temperature controllers are used to cycle fans on and off during the day. Vibrations from the start-up of these fans can cause dirt and microbes trapped inside ductwork to be dislodged and carried into occupied areas.

Another energy conservation effort that may contribute to sick building syndrome is the recirculation of indoor air, at the expense of fresh outdoor air. The 35% of the buildings mentioned above were saving energy by shutting off all the fresh air.

Extremely bad distribution of air throughout the building is common, especially in those systems using multiples of fan coil units mounted throughout the various floors of the building. Local thermostats switch off individual units independently of others and micro-environments are set up. Often it is necessary to ensure that when the heating or cooling is not required, all the fans should be left running to aid circulation throughout the areas concerned.

Variable air volume systems (VAV) using VAV mixing boxes mounted in the ceiling void frequently have louvers opening into the void. When certain temperature conditions are met, the louvers open and return or exhaust air from the void can be induced into the supply air, bypassing the filtration system. We have found fiberglass, asbestos, fungi, and ETS to be recycled throughout an office due to this design.

More and more frequently one finds the following design condition, exhaust fans rated at say 70 to 80% of the supply fans. The supply fans are often automatically throttled back for energy savings, say to 25% of their rated capacity. If the exhaust fan is not adjusted at the same rate the exhaust fan can overpower the supply fan and no fresh air gets into the building. The open fresh air louvers now act as addition exhausts and the whole building runs at negative pressure. When this occurs, unfiltered outside air infiltrates into the building or, worse still, exhaust fumes are sucked up from underground garages.

In addition, as described above, the substitution of low cost, low efficiency filters to reduce pressure drops and save energy seriously reduces the efficiency of building filtration systems, and can lead to serious indoor air quality problems.

## VENTILATION COSTS

Without doubt, the major resistance to increasing ventilation rates has been the cost of such increases. Most companies have incorporated energy management problems and new operating budgets based on saving every energy dollar possible. In fact, the very salaries and bonuses of building engineers or energy managers are dependent on reduced costs. It would be an anathema for them to consider increasing energy usage and cost by increasing ventilation.

However, forward thinking companies should look way beyond the constraints of budgets of the energy managers. Consider the following: the average heating, ventilation, and air conditioning operating costs of a typical 100,000 square foot building in the Washington, D.C. area would be $50,000 per annum. A commendable target for energy saving by saving on ventilation may be say 25% savings, giving a useful $12,500 per annum. Of course, many of you present



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operate buildings many fold larger than 100,000 square feet, so these savings are an attractive goal (see Table 4).

Table 4. Energy Conservation.

Consider a 100,000 square foot building
Typical total utilities cost ($1.25 and $1.75/sq ft)
Average: $1.50/square foot = $150,000 per annum

Typical HVAC fraction (25% to 40%)
Average: 33% = $50,000 per annum

Thus: All energy conservation steps by reducing ventilation, increasing air recirculation, etc. contribute a fraction of $50,000 per 100,000 square foot
Note: a 25% energy savings = $12,500 per year

Now, consider the payroll costs for people in that building. Using typical averages, there are 150 square feet of space per employee, therefore each 100,000 square feet would house 667 people. Supposing we paid these staff only $15,000 per annum for the salary plus payroll costs, the salary bill (667 x $15,000) would be approximately $10,000,000 per annum per 100,000 square feet. Thus, each 1% absenteeism costs $100,000 per annum (see Table 5). Typical absentee rates run at 3 to 7% and no less than 30 to 50% of all absenteeism is estimated to be due to upper respiratory problems. How many of these are due to dusts, bacteria, fungi, fibers, chemicals, ETS, carbon monoxide, oxides of nitrogen, etc., i.e., how many are due to these internal pollutants.

Table 5. Payroll Costs.

Consider 100,000 square feet
Average staffing = 150 square foot/employee
$\frac{100,000}{150}$ square feet = 667 employees

Assume average salary and benefits = $15,000 per annum
667 x $15,000 = $10,000,000 per annum
i.e., each 1% absenteeism costs $100,000 per annum

Note on Average: Upper respiratory complaints = 30 - 50% of all absenteeism

In short, what does it profit a company to save $12,500 in energy savings if that small saving causes potentially hundreds of thousands of dollars in absenteeism, not to mention lost worker efficiency. Small wonder that some European countries, including Denmark, West Germany, and Switzerland have introduced legislation mandating that steps must be taken to prevent the buildup of internal pollutants. The United States is destined to follow that course either by slow evolution or legislation will be precipitated as a result of court actions brought by individuals or by trade unions making the building owners, architects, designers, and operators responsible for the health and welfare of their staff or tenants.

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# PRESENT AND FUTURE OF INDOOR AIR QUALITY

Proceedings of the Brussels Conference,
14–16 February 1989

*Editors:*

**C.J. Bieva, Y. Courtois and M. Govaerts**

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# FOREWORD

Indoor air quality conservation and procedures for the measurement of related potential pollutants, such as radon, asbestos, gases, pesticides, tobacco smoke and bacteria from air conditioning systems, have seen important changes in recent years, while the range and the scope of the studies have continued to expand.

In addition to helping preserve public health, the field of interest is now extending to include such areas as architectural design, ventilation engineering, sociology, psychology and legal aspects. Related analytical techniques like gas chromatography and mass spectroscopy have undergone parallel refinements and their range of application has broadened.

These advances were discussed at the Conference 'Present and Future of Indoor Air Quality', held in Brussels, February, 1989, following symposia on indoor air quality at Essen and Tokyo in 1987 and London in 1988. The sessions were attended by about 200 scientists representing 20 countries. A total of 92 papers and posters were presented covering such topics as pathogenesis and epidemiology, sources of indoor air contamination and risk assessment, chemistry of indoor air related to the outdoor air quality, social and psychological aspects of poor indoor air quality, motivation and attitudes, future guidelines for the improvement of indoor air quality through architectural and ventilation design, and air quality monitoring.

The proceedings include full texts and posters presented during the meeting. The organising committee hopes that they will constitute a useful guide for the improvement of our indoor air quality in the future.

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Present and future of indoor air quality.
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# A DATABASE OF PROBLEM BUILDINGS: LEARNING BY PAST MISTAKES

C.W. COLLETT and E.M. STERLING
Theodor D. Sterling and Associates Ltd., 20-1507 West 12th Avenue, Vancouver, British Columbia, V6J 2E2

T.D. STERLING and J.J. WEINKAM
Faculty of Applied Science, School of Computing Science, Simon Fraser University, Burnaby, British Columbia, V5A 1S6

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## INTRODUCTION

An implicit objective of the designers of modern office buildings is the creation of a "healthy" work environment, one in which building occupants are satisfied with indoor environmental conditions. However, since the 1970's, it has become apparent that occupants of many buildings are not satisfied, frequently reporting a high prevalence of health and comfort complaints which they relate to their work environment. This dissatisfaction has adversely effected productivity through higher absenteeism and increased stress. This type of problem is commonly referred to as the "Sick Building Syndrome". This syndrome was first recognized in Scandinavia in the early 1970's, and has subsequently been widely studied in other countries of Western Europe and in North America (1,2). The most commonly reported symptoms include eye, nose and throat irritation, headache, fatigue, nausea, dizziness and skin rash or itchiness. In addition, occupants of sick buildings often report comfort problems such as a lack of fresh air, stuffiness, inadequate temperature control and unpleasant odours.

A large number of investigations of sick buildings have now been conducted by government and private sector organizations. These past building investigations offer design professionals a vast quantity of valuable information about "mistakes" made in the past. Designers can learn how to create "healthy buildings" in the future from the mistakes that have caused "sick buildings" in the past.

## A DATABASE OF PROBLEM BUILDINGS

An information system, the Building Performance Database (BPD) has been developed as a tool for building professionals and researchers. The BPD currently contains the results of 366

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building investigations conducted by both government agencies (such as NIOSH and Health and Welfare Canada) and private sector organizations. The BPD is an on-line synthesis of a wide range of information collected by the investigators of sick buildings. The database includes bibliographic information about the investigators and their conclusions, architectural and engineering data, and the results of air quality measurements, building system testing and surveys of occupant health and comfort.

BPD is installed on a mainframe computing system in Canada which can be accessed worldwide through existing communications networks. A more complete description of the analytical capabilities of the BPD has been presented elsewhere (3).

## PAST "MISTAKES"

The findings from past investigations can provide useful information to design professionals as they plan future buildings. Knowledge about the causes of past occupant complaints can influence design decisions.

Table One summarizes the conclusions from the 366 reports currently contained in the BPD. Review of the data in BPD shows that in many cases, investigators may identify several factors which they feel had all contributed to the building related problems. Consequently, the table summarizes those factors which have been reported by investigators as contributing to building problems. These factors include ventilation related problems, specific indoor pollutants, stress and ergonomic design. Because investigators have frequently indentified more than one cause in a building, the total number of suspected causes in the table does not equal the number of reports in the BPD. This format differs from tables previously developed by NIOSH and Health and Welfare Canada (4,5,6), which have reported one cause per building (i.e. 400 investigations and 400 suspected causes), which may have oversimplified their results. As the objective of this paper is to provide designers with input into future design decisions, the table provides more detail on specific building problems than those of NIOSH and Health and Welfare Canada.

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TABLE 1

INVESTIGATOR'S CONCLUSIONS FROM REPORTS CONTAINED IN THE BUILDING PERFORMANCE DATABASE

| SUSPECTED CAUSE INDENTIFIED | Number | Percent |
|---|---|---|
| VENTILATION CONTROL PROBLEM | 159 | 39.0% |
| - lack of outside air | | |
| - poor air distribution | | |
| - poor temperature control | | |
| - operational deficiency | | |
| VENTILATION INFILTRATION PROBLEM | 40 | 10.0% |
| - reentry of exhaust fumes | | |
| - outside infiltration | | |
| INDOOR SOURCES | | 28.1% |
| - offgassing from building materials | 27 | |
| - printshops/duplicators | 17 | |
| - microbial | 14 | |
| - smoking | 12 | |
| - fibrous insulation | 8 | |
| - chemical storage | 6 | |
| - cleaning solvents | 6 | |
| - pesticides | 5 | |
| - lighting | 5 | |
| - refrigerant spill | 4 | |
| - carpet shampoo | 4 | |
| - other (boilers,water leaks, etc.) | 7 | |
| STRESS | 12 | 2.9% |
| ERGONOMIC/WORKSTATION DESIGN | 5 | 1.2% |
| UNDETERMINED CAUSE | 42 | 10.2% |
| NO PROBLEM IN BUILDING | 35 | 8.6% |
| TOTAL | 408 | 100.0% |

Ventilation Related Problems

"Ventilation Related" inadequacies are the most commonly reported cause of problems in buildings. A differentiation can be made between problems of "control" and "infiltration".

Ventilation Control Problems. In 39% of the cases contained in BPD, investigators found problems such as inadequate outside air supply, poor air distribution, poor temperature control, a lack of humidification, and various operational deficiencies (such as mechanical failure and poor maintenance practices). All of these causes are "control" problems within the mechanical systems, that

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can often be corrected with fine tuning of the systems. Although not specifically stated in the report conclusions, ventilation control inadequacies probably contribute to other problems in buildings. For example, in those reports where a specific indoor pollutant was cited as the suspected cause, e.g. offgassing of formaldehyde or smoking, the specific pollutant may have been removed from the building if the mechanical systems had been operating effectively.

Ventilation Infiltration Problems. In 10% of the investigations, the cause of building problems was contamination of the outside air supply, resulting from inappropriate location of an outside air intake. Investigators have identified two distinct causes of infiltration problems. First, reentrainment of air exhausted from the building due to the close proximity of air intake and exhaust vents. Second, infiltration from external sources such as automobiles or industry due to improper location of outside air intakes, e.g. at street level on busy downtown streets, adjacent to loading zones or downwind from industrial plants. Designers of future buildings must make certain that outside air intakes are not located such that reentrainment or external infiltration can occur.

Indoor Sources

Specific sources of indoor pollution were cited as the suspected causes of problems in 28.1% of the reports contained in BPD. The specific sources are identified in the table. The most commonly reported sources include offgassing from various building furnishings and finishing materials (20 reports), contamination from print shops, spirit duplicators and blueprint machines (17 reports), and microbial contamination (14 reports). Microbial contamination may partly also be considered "ventilation related", as problems have occurred as a result of the presence of standing water and dampness in HVAC system components. Smoking has been cited as contributory factor to building problems in 12 reports contained in BPD. Other identified sources include pesticides, insulation materials, carpet shampoo and leakage from boilers and water piping.

Stress

In 12 investigations, stress was reported as a contributory factor to the building related problems. In these cases, investigators were unable to determine a physical cause. However,

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negative occupant perceptions about the work environment was cited as the main problem. Investigators of problem buildings must take care not to intensify occupant concerns by their presence in the building and "speculation" about building problems prior to final analysis. Recent research has suggested that the presence of investigators in a building may act as a psychological stressor, intensifying occupant belief that problems exist (7). Such problems can be avoided with carefully designed and executed building evaluations. Standardized protocols for building evaluations have been developed by several researchers (8,9).

Ergonomic Design

An important consideration for interior designers is work station design. Although few investigators of sick buildings appear to have considered ergonomic factors, recent research has suggested that VDT work station design is an important factor in occupant comfort for many white collar workers (10). Poor work station design and/or interior layout wes cited as a suspected cause of building problems in five reports contained in BPD.

Unidentified Causes

In 42 cases, investigators were unable to determine the cause of building related problems. All environmental parameters were "normal", and mechanical systems were operating according to established standards.

No Problem in Building

In 35 cases, investigators concluded that there was no problem in the building under investigation, i.e. no health hazard existed. In most of these cases, investigators were asked to evaluate the potential for building related problems, rather than responding to specific complaints.

DISCUSSION

A vast array of information is now available to building design professionals and researchers, which identifies the probable reasons for occupant health and comfort complaints in modern office buildings. This information has been (and will continue to be) synthesized into a database information system, which may be accessed by building designers and engineers.

Review of the findings of the investigations contained in this

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database suggest that some of the problems that have plagued many sick buildings could have been avoided at the design stage. This is not to say that designers should be blamed for all sick building problems. A building may have been appropriately designed, but later actions by the building users has created the problem. For example as a result of energy conservation measures, alteration or the building without attention to the mechanical systems, overcrowding, poor upkeep and maintenance, or introduction of specific sources of pollution to the building. Whatever the cause of past problems in sick buildings, tools such as the BPD will be valuable in the efforts to design healthier work environments in the future, in which occupants will be more satisfied with their indoor environment and consequently more productive.

One final word of caution. The creation of healthy buildings is not solely a design issue. Once a building has been properly designed, building operators and users should practice a preventative approach to ensure that the building stays healthy thoughout its life. The design stage should be viewed as the first stage of an ongoing process termed "Building Commissioning", which continues to monitor the functional performance of building through construction and occupancy (11,12).

REFERENCES

1. Stolwijk JAJ (1984) In: Berglund B, Lindvall T, Sundall J (eds) Indoor Air '84. Swedish Council for Building Research, Stockholm, Vol 1, pp 23-30

2. Wallace LA (1988) The Sick Building Syndrome: A Review. Proceedings, 81st Annual Meeting of the Air Pollution Control Association, Dallas, Texas

3. Collett CW, Sterling EM, McIntyre ED, Steeves JF, Weinkam JJ (1987) In: Seifert B (ed) Indoor Air '87. Institute for Water, Soil and Air Hygiene, West Berlin, Vol 2, pp 482-486

4. Melius J, Wallingford K, Keenleyside R, Carpenter J (1984) Annals Am Conf Gov Ind Hyg 10:1-10

5. Crandell MS (1988) NIOSH Indoor Air Quality Investigations: 1971 through 1987. Proceedings, 81st Annual Meeting of the Air Pollution Control Association, Dallas, Texas

6. Kirkbride J (1985) Sick Building Syndrome: Causes and Effects. Health and Welfare Canada Report, Ottawa, Canada

2023383830

Source: https://www.industrydocuments.ucsf.edu/docs/zqyx0000

7. Hedge A, Sterling EM, Collett CW, Mueller B, Robson R (1987) In: Seifert B (ed) Indoor Air '87. Institute for Water, Soil and Air Hygiene, West Berlin, Vol 2, pp 552-556

8. Woods JE, Morey PR, Rask DR (1987) Indoor Air Quality Diagnostics: Qualitative and Quantitative Procedures to Inprove Environmental Conditions. Presentation, Symposium on Design and Protocol for Monitoring Indoor Air Quality. ASTM Symposium, Cincinnati, Ohio

9. Sterling EM, Collett CW, Meredith J (1987) A Five Phased Strategy for Diagnosing Air Quality and Related Ventilation Problems in Commercial Buildings. Proceedings, 80th Annual Meeting of the Air Pollution Control Association, New York

10. Smith MJ, Carayon P, Miezio K (1987) In: Knave B, Wideback PG (eds) Work with Display Units '86. Elsevier Science, Amsterdam, pp 695-712

11. ASHRAE (1988) Proposed Guideline for Commissioning Building Mechanical Systems (Public Review Draft). ASHRAE, Atlanta, Georgia

12. Sterling EM (1988) ASHRAE Transactions, 95(1); CH-89-13-5

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# AN INDOOR AIR QUALITY SURVEY OF TWENTY-SIX SWISS OFFICE BUILDINGS

Simon Turner and Peter W.H. Binnie
Healthy Buildings International Inc. (HBI)
10378 Democracy Lane
Fairfax, Virginia 22030
U.S.A.

In an effort to characterize the major factors influencing air quality in buildings in Switzerland, 26 representative buildings were selected for this study. Each building was subjected to the same indoor air quality survey methodology. The most significant cause of air quality problems was found to be poor ventilation, followed by inadequate filtration and poor hygiene. Control of Legionella bacteria and asbestos-containing materials may also require high priority in order to prevent immediate and long term hazards to building occupants.

## INTRODUCTION

There is a continuing requirement for air quality data on buildings not classified as "sick" which are representative of buildings as a whole within a specific region or country. Currently, data on levels of many common indoor pollutants in Switzerland are sparse. In addition, it has been shown in the U.S. (1,2,3) that maintenance activities and the condition of air handling equipment can have a profound affect on indoor air quality. Information on these factors in Switzerland is equally hard to find.

This survey selected and evaluated a representative group of 26 commercial office buildings comprising a total of approximately 102,300 square meters of office space in 20 cities in Switzerland. The objective of this survey was to provide contributory data for future mitigation policies, as well as to help set guidelines for suitable ventilation rates and filtration standards in particular. These studies were carried out from the 7th of February to the 15th of March 1989. The buildings selected for this survey were of a wide variety in terms of their size, construction and use. However, a standard methodology to investigate each building was applied.

## METHODOLOGY

Although a standard approach was used to survey each building, it required flexibility to cope with different types of buildings examined.

### Initial Walk Through

Since one of the objectives of this survey was to assess maintenance standards, each study commenced with an interview with the personnel responsible for maintenance of the building. Questions were designed to elicit operative details such as system on/off times; fresh air, return air and exhaust settings; scheduled maintenance routines; and complaint areas, if any, but did not include questioning of the occupants. There was a walk through of each building to

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identify obvious building configurations or design features which could influence air quality in the occupied areas. This was followed by a complete visual inspection of the internals of the building's ventilation system, if any. A visual inspection was also made of the internals of the main air supply ductwork leaving each air handling unit. Where necessary, access was gained to this ductwork by the installation of a small access port and the insertion of a fiber optic borescope.

Qualitative Sampling

In each air handling unit and main air supply duct, a series of samples were also collected on cellulose ester filters for light microscopy analysis. Surface microbe samples were collected on Random Organism Detection and Counting (RODAC) agar plates, to be subsequently incubated, counted, and identified.

A laser particle counter with a size-selective inlet for sampling particles with an aerodynamic diameter of 0.5 microns and above, was used to count particles inside the ductwork. At least two points were sampled inside each major run of ductwork. This qualitative information on the building, along with the location of the samples and the building engineer questionnaire, was prepared on a set of standard field notes to ensure consistency. In the case of buildings not equipped with forced air ventilation systems, this walk through and sampling phase was obviously more limited in scope.

Quantitative Air Sampling

A set of locations were identified in each building to be used for quantitative airborne sampling. These locations were spread evenly throughout the study area of each building with a minimum of two locations per floor, as well as an outdoor control sample point. The following parameters were measured at each location where relevant and appropriate:

- Respirable airborne particle counts, using a piezoelectric microbalance;
- Carbon dioxide levels, using a non-dispersive infrared absorption portable gas analyzer;
- Carbon monoxide concentrations, using a controlled potential electrolysis detector;
- Airborne nicotine (after Ogden et al (4)), with a personal universal flow sampling pump;
- Temperature, using a miniature platinum Pt 100 resistance sensor; and
- Relative humidity, using a chromed layered capacitative electrode.

The following parameters were measured in at least two selected locations in each building:

- Miscellaneous gases, using Gastec calibrated detector tubes
- Airborne microbial counts, using a centrifugal air sampler employing impaction onto an agar lined drum;
- Formaldehyde, using midget impingers containing sodium bisulphite, followed by spectrophotometric analysis;
- Radon gas in basement areas, using Track-Etch radon detectors;
- A range of volatile organic compounds, using a GC/MS in one or two locations per building, plus an outside control for each city;
- Bulk asbestos analysis of any materials in the air stream of the ventilation system, or exposed to the building occupants, which were suspected of containing asbestos fibers;
- Sampling and counting of airborne asbestos fibers;
- Sampling of selected water sources, and analysis for the presence of *Legionella pneumophila*, along with a hazard assessment of the water source for possible future contamination and dissemination of this organism.

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## RESULTS AND DISCUSSION

This survey yielded a large amount of data which can be broadly classified as either quantitative analytical data or more empirical assessments of the condition of the air handling systems. The quantitative data is shown in Tables I, II and III, with the qualitative data assessed and ranked on Figure 1.

Nitrogen dioxide, lower and higher range hydrocarbons, ozone, ammonia, and sulphur dioxide were not found above the detection limit of the method used and are, therefore, not included in these tables.

**TABLE I. MECHANICALLY VENTILATED AREAS**

| Building Area | $CO_2$ (ppm) am | $CO_2$ (ppm) pm | CO (ppm) am | CO (ppm) pm | RSP ($\mu gm^{-3}$) am | RSP ($\mu gm^{-3}$) pm | Total HCHO (ppm) | VOCs ($\mu gm^{-3}$) | Nicotine ($\mu gm^{-3}$) | Radon ($Bqm^{-3}$) | Airborne microbes ($cfum^{-3}$) |
|---|---|---|---|---|---|---|---|---|---|---|---|
| A (all) | 628 | 545 | 4.3 | 4.1 | 41 | 35 | 0.03 | 1161 | 6.6 | -- | 238 |
| C (all) | 408 | 408 | 2.5 | 2.7 | 53 | 53 | 0.01 | 64 | <DL | 40 | 340 |
| F (all) | 454 | 587 | 3.6 | 3.7 | 13 | 19 | 0.01 | 2030 | 4.0 | 18 | 111 |
| I (all) | 496 | 442 | 4.3 | 4.4 | 29 | 35 | 0.04 | 515 | 2.9 | 29 | 170 |
| R (all) | 528 | 492 | 2.5 | 0.8 | 20 | 28 | <DL | -- | -- | 33 | 374 |
| Y (all) | 473 | 491 | 3.5 | 3.3 | 14 | 12 | 0.02 | 276 | 3.5 | 81 | 600 |
| Z (basement) | 500 | 600 | 3.0 | 2.5 | 15 | 10 | 0.03 | 772 | -- | 66 | 1000 |
| MEAN | 498 | 509 | 3.4 | 3.1 | 26 | 27 | 0.02 | 900 | 3.4 | 44 | 405 |

**TABLE II. NATURALLY VENTILATED AREAS**

| Building Area | $CO_2$ (ppm) am | $CO_2$ (ppm) pm | CO (ppm) am | CO (ppm) pm | RSP ($\mu gm^{-3}$) am | RSP ($\mu gm^{-3}$) pm | HCHO (ppm) | Total VOCs ($\mu gm^{-3}$) | Nicotine ($\mu gm^{-3}$) | Radon ($Bqm^{-3}$) | Airborne microbes ($cfum^{-3}$) |
|---|---|---|---|---|---|---|---|---|---|---|---|
| D (B-2F and 5F-8F | 523 | 564 | 2.4 | 3.4 | 21 | 36 | 0.06 | -- | -- | 77 | 334 |
| E (all but lab | 600 | 622 | 2.7 | 6.7 | 92 | 56 | 0.06 | 824 | <DL | 62 | 768 |
| G (all but reception | 858 | 642 | 6.2 | 4.2 | 118 | 357 | 0.06 | 991 | 23.4 | 37 | 775 |
| H (all but computer/conference rooms) | 669 | 572 | 2.1 | 3.5 | 14 | 16 | 0.12 | 2693 | 3.9 | 44 | 116 |
| J (all) | 679 | 871 | 3.4 | 3.6 | 29 | 34 | 0.20 | 1206 | 41.9 | 29 | 533 |
| K (all) | 817 | 817 | 1.5 | 1.8 | 98 | 87 | 0.04 | 2859 | 18.3 | 48 | 268 |
| L (all) | 938 | 588 | 2.5 | 2.0 | 31 | 14 | 0.07 | 2033 | 2.8 | 59 | 157 |
| M (total study area) | 733 | 544 | 2.0 | 2.0 | 49 | 30 | 0.01 | 562 | 17.8 | 23 | 366 |
| N (all) | 746 | 750 | 2.0 | 2.0 | 16 | 16 | 0.02 | 414 | 15.1 | 275 | 254 |
| O (complete study area except 3F conf, lounge, and 2F lounge) | 692 | 713 | 3.3 | 2.4 | 33 | -- | 0.02 | 935 | -- | 29 | 273 |
| P (all) | 670 | 895 | 2.4 | 2.0 | 25 | 33 | 0.02 | 702 | <DL | 37 | 171 |
| T (all) | 731 | 613 | 2.5 | 2.0 | 32 | 24 | 0.03 | 146 | 9.0 | 209 | 156 |
| U (Ground floor offices, basement) | 600 | 475 | 3.0 | 2.5 | 100 | 85 | 0.04 | -- | -- | 37 | 500 |
| V (all) | 683 | 692 | 3.5 | 2.5 | 33 | 15 | 0.04 | 84 | 5.3 | 92 | 325 |
| W (all) | 556 | 600 | 2.7 | 2.6 | 46 | 52 | 0.02 | 910 | <DL | 532 | 372 |
| X (all) | 900 | 720 | 2.4 | 2.4 | 144 | 78 | 0.03 | 138 | <DL | 14,641 | 38 |
| Z (all but basement) | 575 | 658 | 2.3 | 2.3 | 45 | 78 | 0.02 | -- | 3.0 | -- | 454 |
| MEAN | 704 | 667 | 2.8 | 2.8 | 55 | 63 | 0.05 | 1036 | 10.0 | 106* | 345 |

*This mean figure does not include the result from Building X.



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TABLE III. MIXED VENTILATED AREAS

| Building Area | $CO_2$ (ppm) am | $CO_2$ (ppm) pm | CO (ppm) am | CO (ppm) pm | RSP ($\mu gm^{-3}$) am | RSP ($\mu gm^{-3}$) pm | HCHO (ppm) | Total VOCs ($\mu gm^{-3}$) | Nicotine ($\mu gm^{-3}$) | Radon ($Bqm^{-3}$) | Airborne microbes ($cfum^{-3}$) |
|---|---|---|---|---|---|---|---|---|---|---|---|
| B (all) | 579 | 561 | 5.4 | 4.6 | 19 | 16 | 0.04 | 2420 | 25.7 | 37 | 377 |
| D (3F-4F) | 460 | 450 | 3.0 | 3.4 | 12 | 14 | 0.01 | 1366 | <DL | -- | 271 |
| E (GF lab) | 1250 | 1850 | 3.0 | 3.0 | 80 | 130 | -- | 599 | -- | -- | 259 |
| G (GF reception | 350 | 400 | 3.0 | 2.0 | 40 | 80 | -- | -- | <DL | -- | 775 |
| H (computer/ conference) | 550 | 700 | 2.0 | 4.0 | 20 | 10 | -- | -- | -- | -- | 13 |
| O (3F conf. lounge and 2F lounge) | 508 | 617 | 3.3 | 2.7 | 25 | -- | -- | -- | 2.7 | -- | 516 |
| Q (all) | 620 | 573 | 2.4 | 2.7 | 55 | 13 | 0.03 | 329 | 2.1 | 33 | 533 |
| S (all) | 750 | 517 | 3.2 | 2.8 | 43 | 42 | 0.03 | 321 | 1.4 | 77 | 554 |
| U (1F, 3F, GF cafe) | 550 | 558 | 2.7 | 2.8 | 70 | 57 | -- | 1070 | 1.1 | -- | 481 |
| MEAN | 624 | 692 | 3.1 | 3.1 | 40 | 46 | 0.03 | 1018 | 4.7 | 48 | 420 |

| | | | |
|---|---|---|---|
| CO | carbon monoxide | $CO_2$ | carbon dioxide |
| RSP | respirable size particles | HCHO | formaldehyde |
| VOCs | volatile organic compounds | $\mu gm^{-3}$ | microgram/cubic meter |
| ppm | parts per million | DL | detection limit |
| -- | not tested for | $Bqm^{-3}$ | Becquerels/cubic meter |
| $cfum^{-3}$ | colony forming units/cubic meter | | |

These 26 buildings varied widely, and as a result, there were many items found which were unique to a particular building. Specific problems included HVAC outside air intakes directly adjacent to parking lots. This type of problem is reflected in the "building configuration" section of Figure 1. Another problem unique to a building was an excessively high radon concentration in an area of Building X which required immediate attention. There were, however, some other more pervasive factors which were common to at least a sub-set of the buildings.

Since this survey was made in winter, most operable windows remained closed. As a result, many of the buildings that were not equipped with forced ventilation systems became somewhat stuffy, and this was reflected in elevated carbon dioxide concentrations, either throughout the building, or in certain pockets. Table III shows carbon dioxide levels overall in the naturally ventilated buildings to be slightly lower in the afternoon -- this coincides with observations that windows were more often open in the warmer afternoon hours than in the colder mornings. Table III also shows that most indoor pollutants measured were marginally higher in the naturally ventilated areas. An exception is carbon monoxide, which was generally lower and this may be due to the presence of underground parking garages under many of the sealed, mechanically ventilated buildings.

Low ventilation rates can be seen to affect more than half of these 26 buildings (Figure 1) and may be indicative of a general trend. Since the majority of underventilated buildings were older structures with little or no mechanical ventilation system, it demonstrates that indoor air quality problems are not limited to U.S. style "high tech" sealed buildings, but are just as likely in older buildings (at least in seasons of adverse weather).



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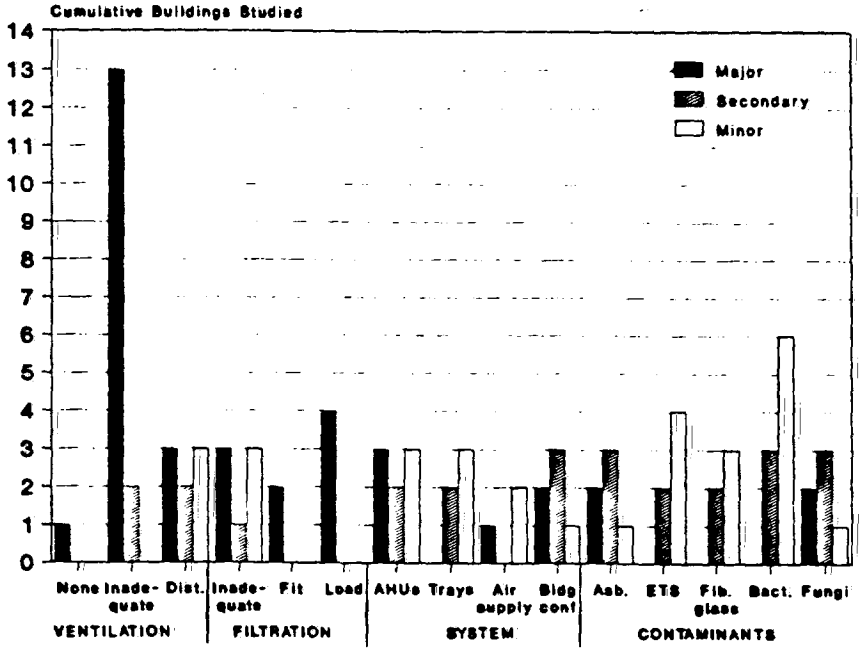
Figure 1. Indoor Air Quality Problems from 26 Swiss Buildings




None - no fresh air
Inadequate - limited fresh air
Dist. - poor fresh air distribution
AHUs - dirty air handling units
Trays - blocked or dirty condensate trays
Air supply - dirty air supply ducts
Bldg Conf - building design or configuration problems

Inadequate - low efficiency filters
Fit - poor fitting filter
Load - excessively dirty filters
Asb. - presence of asbestos
ETS - environmental tobacco smoke
Fib. glass - loose in air supply system
Bacteria - presence of infectious or allergenic bacteria
Fungi - presence of infectious or allergeni fungi

Overall, dust levels were higher in the naturally ventilated buildings not equipped with a filtration system. This is not surprising since dusts generated by occupant activities are more likely to be suspended in the room air for long periods instead of being drawn into a return system. In the more sophisticated buildings, a number of filter systems were found to be subjected to poor maintenance -- most commonly the selection of filters which are likely to rate less than 10% efficient in the respirable size range. Most commercial systems should be fitted with filters at least 20% efficient in this size range. A minority of these filters were poorly fitted, allowing air bypass, and four buildings were found to have filters which were excessively loaded. We still require a standard test which evaluates the ability of the filter to remove sub-micron size particles since these are the ones that penetrate deep into the respiratory system. We currently do not have such a test which is applied routinely to commercially available filters.

Due to poor maintenance, heavy dirt created problems in most of the air handling units inspected. Condensate trays and air supply ducts were also found to be

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loaded with various levels of dirt, slime or scale. Sampling for *Legionella pneumophila* in building water using the Legionella Rapid Assay method, revealed trace levels of the organism in five buildings, and strongly positive results in three buildings. These made up the majority of the bacterial problems found in this survey and demonstrate that this organism may be quite widespread in cooling and humidifying systems in Swiss buildings. In all twenty-six buildings, airborne microbial samples yielded wide ranges of fungal species. These were generally similar to outdoor air sample results. Better attention to hygiene of air handling systems may be one of the more effective ways of reducing occupant exposure to irritants in many Swiss buildings.

A minority of buildings exhibited poor use of fibrous glass, creating the potential for release of fibers either into the air stream of the ventilation systems, or directly into the room air. This is a maintenance item which is relatively simple to correct. More complex problems are raised by asbestos containing materials which were found to be a significant problem in two of the buildings examined, and secondary or minor problems in a further four. The control of fiber release from asbestos containing products in Swiss buildings may be a topic which requires significant educational effort in the future.

Environmental tobacco smoke was found to be a secondary or a minor irritant in a total of six of these buildings, usually associated with pockets of poor ventilation. Ventilation rates which maintained carbon dioxide levels consistently below 800 ppm, resulted in low levels of ETS, both as measured by nicotine and RSP levels.

The most room for improvement in these buildings was found with ventilation rates, which were inconsistent, and, in mechanically ventilated buildings, with overall levels of maintenance, especially with regard to filters and cleaning schedules. In particular, there is a need for a standard filter testing method for respirable dust removal. Furthermore, if these buildings are representative of many Swiss buildings, control of *Legionella* bacterium in cooling systems, improvement of hygiene of air handling units to maintain low levels of bacteria and fungi, and abatement of asbestos containing materials in exposed areas may need to be given high priorities in order to prevent immediate and long term hazards to building occupants.

**ACKNOWLEDGEMENTS**

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**REFERENCES**

1. Robertson G (1988) Source, Nature, and Symptomology of Indoor Air Pollutants, Indoor and Ambient Air Quality. Selper, London. p. 311-19.

2. Collett CW, Sterling EM, Arundel AV (1988) Changing Sick Buildings into Healthy Buildings: Improving the Ventilation System, Proc. Healthy Buildings 88. Vol. 3, Stockholm, Sweden.

3. Morey PR, Rundus RE (1984) HVAC System Operational Performance Affect Airborne Fungal Levels in Occupied Spaces, Proc 3rd Int. Conf. on Indoor Air Quality and Climate. Vol. 3, Stockholm, Sweden.

4. Ogden NW, Eudy LW, Heavner DL, Conrad Jr FW, Green CR (1986) Improved Gas Chromatographic Quantitation of Trace Levels of Environmental Nicotine, 40th Tobacco Chemists' Research Conference, Knoxville, TN.

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# Indoor Air Quality

## Symposium

December 6 - 7 , 1988
**San Carlos de Bariloche**
**Argentina**

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# THE EPIDEMIOLOGY OF "SICK PUBLIC BUILDINGS"

*by THEODOR STERLING and S.KLEVEN*

Dr. Sterling is a Professor in the Faculty of Applied Science at Simon Fraser University, Burnaby, British Columbia, Canada. He is a Fellow of the American College of Epidemiology, the Canadian Committee of Scientists and Scholars, the American Association for Achievement of Science and the American Statistical Association.
Ms. Kleven received her Master of Science in Kinesiology in 1986. She is a Research Analyst with Theodor D. Sterling Limited.

## ABSTRACT

The indoor environment of modern buildings, especially those designed for commercial and administrative uses, constitute a unique ecological niche with its own biochemical environment, fauna and flora. Sophisticated construction methods, new materials and machinery required to maintain the indoor environment of these enclosed structures produce a large number of chemical by-products and permit the growth of many different microorganisms. Because modern office buildings are sealed, the regulation of humidification and temperature of ducted air presents a dilemma, since different species of microorganisms flourish at different combinations of humidity and temperature. If the indoor environment of modern office buildings is not properly maintained, the environment may become toxic to the occupants' health. Such buildings are classified as Sick Buildings. A review of the epidemiology of building illness is presented. The etiology of occupant

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illnesses, sources of toxic substances, and possible methods of maintaining a safe indoor environment are described.

## INTRODUCTION

Disease has always been strongly associated with the type and use of structures humans occupy. Food, drink, waste products and the presence of any other organic substances (including animals) harbor infectious organisms. With the discoveries of the vectors by which infectious organisms invade a human host have come innovations in building technology and mechanical appliances that cook, store and preserve food, eliminate human waste, and help keep the indoor environment of a building reasonably free of harmful infectious agents. In addition, although not recognized as a source for disease, are toxic chemicals found indoors. The link between the by-products of combustion of biomass materials burned indoors (such as wood, coal, and later on, kerosene and natural gas) and subsequent chronic disease was not recognized until relatively recently. In general, while the type of structure in which people lived and worked may have had a profound influence on their health, specific antecedents of disease were not so much related to a structure of a building as to a specific point source within the structure (e.g., wastes, storage of food, fires used for heating or cooking).

At the start of the twentieth century, a new type of structure became prominent; that of a large public building devoted to commercial or administrative uses. As these buildings grew in size they created a uniquely new environment plagued with problems, primarily because of the great volume of air they enclosed. In addition, the by-products of the machinery required to make these buildings function also created problems. The requirements of air intake, heating, cooling and distribution, and elimination of by-products of human occupation and machinery created needs for massive ducting throughout the full height of the structures, which led to demands for building designs that were large enough to accommodate the machinery necessary to make human occupation possible. (These included the seldom mentioned flush toilets, without which, skyscrapers would be uninhabitable.) However, the main criteria of these early buildings was not so much to eliminate pollutants created inside the building but to prevent infiltration of pollutants from outside. This was especially true for soot from burning coal



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used for industrial plants and residences. Initially, attempts were made to regulate the indoor climate by warming and cleaning intake air and then ducting it throughout the building. Outstanding examples of this new technology are the Royal Victoria Hospital in Belfast, built in 1903, and the Larkin Administration Building in Buffalo, New York, built in 1906. From these pioneering efforts came the idea that to condition the indoor environment of a large building and to remove some of the sources of discomforts coming from outdoors, well tempered air must be provided indoors. These initial attempts were further supported by the desire to eliminate the wells for lighting and ventilating the inside of buildings. These wells occupied space that could more profitably be used in commercial buildings for additional office space.

It took the first half of the twentieth century to learn how to supply power to, and maintain these large office buildings. However, starting in the late 1960's, the buildings in the commercial districts of modern cities began to change. New buildings were erected that were sealed structures of great height that relied entirely on mechanical means to regulate their indoor environments. Buildings that used operable windows for ventilation and circulating steam or hot water for heat were replaced by structures with ducted air that was centrally heated or cooled. Advancements in chemistry made possible the manufacturing of entirely new fabrics that were used to cover floors and walls, and construct furniture. Formaldehyde resins were used extensively for particle boards from which furniture or wall panels were constructed and in glues to affix carpets to the floors. A variety of machines were introduced, of which perhaps the most important, from an epidemiological perspective, are the photocopier and certain types of air conditioners. To clean the vast floor areas various carpet shampoos and industrial cleaners were used that sometimes left toxic residues. Filters were used to clean the air circulated by means of the ducting systems. At the same time, however, these filters and duct systems became breeding grounds for various types of organisms (as were some carpets). In short, the modern buildings provided conditions to create a complex ecological niche that became a possible source for human disease.

## CAUSES OF BUILDING ILLNESS

Since the early 1970's, occupants of hundreds of modern sealed office



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buildings throughout North America have reported similar health and comfort complaints *(Berglund, 1984)*. These buildings are commonly referred to as "sick buildings" and the epidemic of complaints by the occupants of these buildings has been defined by the World Health Organization as "Sick Building Syndrome" (*WHO*, 1982). Sick buildings are identified by a very high prevalence of specific health symptoms among the occupants which include: headache, eye problems (irritated, sore, dry, itching, or watering eyes), nasal problems (stuffy, runny, or irritated nose), throat problems (dry, sore, or irritated throat), chest problems (chest tightness, difficulty breathing), fatigue and lethargy (including sleepiness and weakness), skin abnormalities (dry, itchy, or irritated skin), and problems maintaining concentration at work (*Sterling, E*, 1983; *Hedge*, 1984; *Finnigan*, 1984; *Robertson*, 1985). In four studies the incidence of "building illness" was found to be at least twice that in naturally ventilated buildings (*Turiel*, 1983; *Hedge*, 1984; *Finnigan*, 1984; *Robertson*, 1985). It has also been suggested that other symptoms, such as skin rashes/irritation/dryness, nausea, dizziness, and respiratory problems (wheeze, shortness of breath, chest tightness) are characteristically more prevalent in sick buildings (*WHO*, 1983; *Stolwijk*, 1974; *Hawkins*, 1985). Finally, there have been complaints in some buildings of increased spontaneous abortions. However, most of these complaints have been anecdotal, and because of the difficulty of counting them, such abortions have not been verified.

The effect of sick buildings on occupant health is demonstrated by the study of *Sterling, E. and Sterling, T.* (1983) of a group of office workers who moved from an old fashioned building with window ventilation and lighting to a modern sealed structure. Absentee data were supplied for a period of one year before and seven months after the study group moved into the new building. The percent of absent days was calculated for all staff members. The study group served as its own control, making possible the comparison of absentee prevalence before and after the move to the new enclosed structure. Figure 1 shows the percentage of days absent for the study group beginning in August 1978 and ending in February 1980. The vertical solid line divides the graph into two parts, before and after the move. There is no trend in absenteeism before the move. Most weekly absences were below 3% and there were no absences at all for 40% of the weeks. The dotted line is the line of best fit resulting from fitting a linear equation to the data for the period of time prior to the move into the study building. The slope of this line is not significantly different from zero. The coefficient of determination



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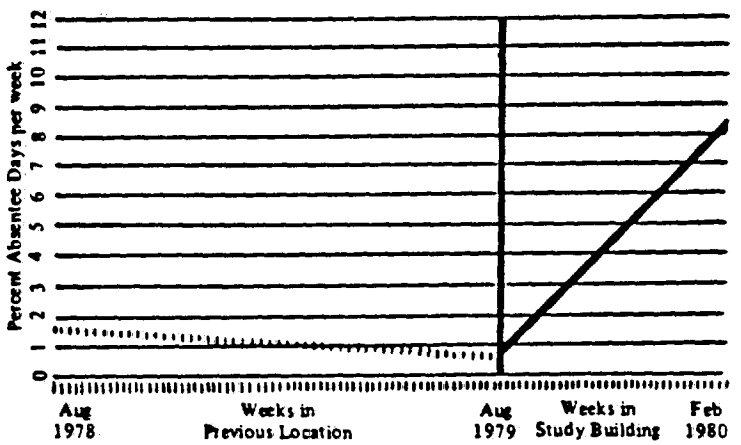
Figure 1. Absentee rate scattergram pre- and post- occupancy of the study building




From Sterling and Sterling (1983).

($r^2$) between absentee rate and time is 0.03 and the correlation coefficient (r) is -0.17, indicating that no increasing (or significantly decreasing) trend is apparent.

The solid line is the line of best fit of the data for the period of time after the move. The positive slope of 2.66 approximates the linear trend of absentee rates per week in the study building during initial occupancy. The $r^2$ in this case is 0.53 and r is .73 ($p < .01$). This significant trend clearly shows that absenteeism was increasing after the move into the new building.

Figure 2 details the striking difference in specific complaints in a cross group comparison between a study and control group. The control group oc-

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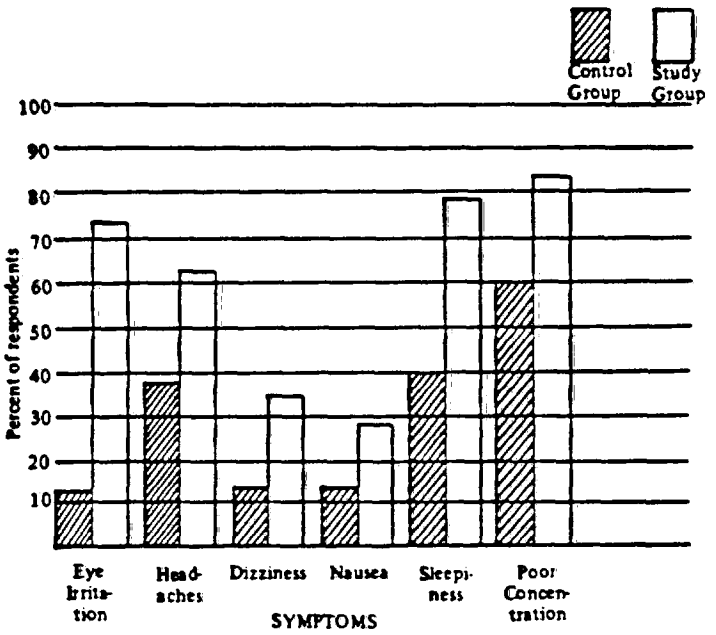
Figure 2. Symptom distribution by group.




From Sterling and Sterling (1983).

cupied a building with operable windows for ventilation and hot water for radiant heat.

## SPECIFIC CAUSES OF BUILDING ILLNESS

A number of studies of sick buildings have been able to isolate instances where the cause of health symptoms could be isolated and eliminated by



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taking appropriate measures. Such examples contribute important information of how to prevent building wide epidemics.

In 1982 my associates and I started to create an archive of reports of studies of sick buildings (*Collett*, 1987). This archive now contains reports of almost 500 building studies conducted in North America (the majority are located in the U.S). These studies were completed by industrial hygienists and investigators working for the National Institute of Occupational Safety and Health (*NIOSH*) of the United States, the U.S. Center for Disease Control, various municipal and state agencies, universities and other research centers.

Here are some examples identified as contributing causes of building illness:

**Formaldehyde:** From insulation (*Gunter*, 1981; *Fannick*, 1981) and offgasing from materials containing formaldehyde resins (*Chrostek*, 1981; *Konopinski, 1980; Coy, 1982).*

**Industrial cleaners:** From the residues of industrial carpet shampoos (*Kreis*, 1981; *Robertson*, 1980).

**Solvents:** From fresh ink on checks in poorly ventilated areas (*Thoburn*, 1981) and printing solvents (*Gunter*, 1981).

**Perchloroethylene:** From the duct work of a dry cleaner. The gas travelled above the ceiling to other stores in the same complex (*Thoburn*, 1981).

**Fiber Glass:** Disseminated through the ventilation system (*Kreis*, 1981).

**Ozone:** (*Shoemaker*, 1977; *Gunter*, 1981).

**Automobile exhaust:** Entered through the ventilation inlets (*Nudelman*, 1979; *Leiderman*, 1982).

**Photocopiers:** From a hot oil process, use of methyl alcohol (*Prior*, 1981; *Apol*, 1981; *Fannick*, 1980; *Chrostek*, 1981).

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The causes of sick building syndrome are very often unclear. Usually, however, the health symptoms in such buldings can be resolved by increasing the fresh air supply (*Salisbury*, 1981; *McManus*, 1985; *Chio*, 1985).

A summary of what is known about specific causes of sick building syndrome was provided by the U.S. National Center for Occupational Safety and Health (*NIOSH*) in 1984. The findings from 203 of NIOSH's investigations undertaken up to the end of 1983 were reviewed and tabulated by the Health Hazards Evaluation Branch of NIOSH (*Melius*, 1984). Of these 203 studies, 17.7% attributed the health symptoms to indoor air contamination (e.g., from sources such as industrial cleansers or photocopiers), 10.3% to outdoor air contamination (e.g., from misplaced air intake vents that would draw gasoline fumes from garages or bus stations), 3.4% to building contamination (e.g., offgasing of formaldehyde from wall panels, glues and resins in carpets, and furniture made from particle board), 40.3% to inadequate ventilation, 3% to hypersensitivity pneumonitis (often caused by the use of humidifiers), 2% to cigarette smoking, 4.4% to humidity, and 1% to noise and lighting. The remaining 10% of the studies did not find a cause for health symptoms. A review by Health and Welfare Canada (*HWC*) of 94 building studies gave similar results (*Kirkbride*, 1985). In this review, 68% of the studies attributed the cause of health symptoms to inadequate ventilation (e.g., poor air circulation, inadequate fresh air intake, and poor temperature and humidity control) 10% to outdoor contaminants (e.g., motor vehicle exhaust entering the building), 5% to indoor contaminants (e.g., photocopy machines and tobacco smoke; the report does not state to what extent the cause of building illness was due to the use of copy machines or cigarette smoke), 2% to building gases (e.g., formaldehyde, organic glues and adhesives), and the remaining 15% to unknown sources. (Because of present concerns with smoking, all NIOSH and HWC studies paid special attention to smoking as a possible cause of sick buildings. It is of interest, therefore, that smoking was said to be a major cause of complaint only in 2% to 5% of the sick building investigations. Removing the smoker entirely, then, may not affect health and comfort problems in 95% to 98% of sick buildings.)

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## BIOCHEMICAL SUBSTANCES IN MODERN BUILDINGS

A wide range of substances have been measured inside modern office buildings. Some of these substances are known, and others are thought to be associated with various diseases. A summary of the measured substances was compiled by the U.S. Environmental Protection Agency and published in the Environment Protection Agency Indoor Air Quality Implementation Plan (1987) under the title "Common Indoor Pollutants, Their Sources and Known Health Effects" (*EPA*, 1987). (Because of its importance, this list is included as an APPENDIX.)

In summary, the EPA table lists 73 common indoor pollutants that are known to be toxic, although not necessarily at the level of concentration found in most buildings. Of these 73 substances, 38 are attributable to building components and appliances, 26 by the evaporation of drinking water, 17 by outdoor air, 16 by products used for cleaning and maintenance, 12 by tobacco smoke, and 9 by vehicles exhaust trapped in garages. Tobacco smoke was singled out by the Environmental Protection Agency because of its visibility. However, with the exception of nicotine and nicotine derivatives, almost all of the substances from tobacco smoke are also derived from a number of other sources. For example, carbon monoxide (CO), besides being present in tobacco smoke, is usually present in much greater quantities, or produced by, HVAC systems, garages, outdoor air and gas stoves; styrine also comes from furnishings and drinking water; benzene from garages and drinking water; methyl chloride from drinking water, pyridine from outdoor air; acetone from cleaners, waxes, adhesives, cosmetics and outdoor air; acroline from the combustion of various products (as are other substances such as formaldehyde or respirable particles); benzo(a)pyrene from automobile exhaust and drinking water; and analine is a by-product from various activities. Sources, other than tobacco, are not given for methylamine and hydrazine. (This may be a result of too few measurements available for these two substances, rather than a real absence of these substances from the combustion of materials other than tobacco.)

In summary, the EPA list demonstrates that there are multiple sources for many chemicals and substances known, or thought, to be toxic. Because

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there are so many alternative sources for most substances found in a building, the best method to deal with indoor exposure to biochemicals is to dilute them by adding an adequate amount of fresh air.

## THE INDOOR ECOLOGY

A general rule of ecology is that every spot capable of supporting life will be occupied by some form of life. This is just as true for the indoor environment as it is for the Arctic Tundra. Countless substances, necessary to sustain life, are found indoors, especially in the ducts and ventilation system of a building and in all places where moisture may gather. Dusts, much of which contain both the staff of life and microbial and viral life itself, are brought into buildings through fresh air intake and on the clothing, hair and skin of building occupants and visitors. Some of the microorganisms can cause violent physiological reactions in humans.

Perhaps the best known example of the possible virulence of indoor microbial life is Legionnaires' Disease. Legionnaires' Disease, named after an explosive outbreak of pneumonia in a group of Legionnaires attending an American Legion convention in Philadelphia in 1976, is a pneumonia-like reaction to the Legionella Pneumonphilia bacterium. The bacterium requires the presence of certain algae, which usually grow in cooling towers at specific temperatures, for growth (*Fraser*, 1977). Pontiac Fever, named after a health department building in Pontiac, Michigan, U.S., in which the syndrome was first described, is a strain of Legionnaires' Disease (*Kaufman*, 1981). Recurrences of Legionnaires' Disease and Pontiac Fever are documented from time to time, very often in hospitals, and are always connected with poor maintenance of cooling towers (*Friedman*, 1987; *Garbe*, 1985; *Conwell*, 1982; *Kaufmann*, 1981; *Fisher-Hoch*, 1981).

Legionnaires' Disease, however, is just the tip of the iceberg. There are many places in a building in which infectious organisms (bacteria, viruses, fungi, mites) and pollen may flourish. As an example, my associates and I reported allergic and infectious reactions caused by humidifier contaminants (*Arundel*, 1986, see TABLE 1). In general, organic materials will flourish at all levels of relative humidity. Figure 3 describes the relationship between

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**TABLE 1. REPORTS OF ALLERGIES CAUSED BY HUMIDIFIER CONTAMINANTS**

| Subjects | Diagnosis | Contaminant | Confirmation |
|---|---|---|---|
| 11 office workers | Fever, malaise, chest tightness, polyuria | Acanthamoeba spp. | Symptoms disappeared 4 weeks after the humidification system corrected |
| 26 office workers | Fever, chills, cough, dyspnea | Unknown, possibly protozoa | No symptoms 5 months after humidification system removed |
| 24 factory workers | Extrinsic allergic alveolitis | Phialophora spp., Cephalosporium, Fusarium, Gliomastix | Precipitins to humidifier water, symptoms ceased after alteration to system |
| 20 factory workers | Fever, chills, dyspnea | Pseudomonas endotoxins in humidifier | Not stated |
| 3 housewives | Recurrent acute interstitial lung disease | Thermoactinomyces in home humidifier | Positive bronchial challenge to hemophile |
| 1 male | Recurrent pneumonia | Fungi and bacteria in humidifier | Challenge with vaporizer aerosol positive, specific agent unidentified |
| 1 female | Recurrent hypersensitivity pneumonitis | Thermotolerant bacteria in home humidifier | Positive bronchial challenge, positive serum |
| 1 female | Hypersensitivity pneumonitis | Unknown organisms in home humidifier | Positive pulmonary challenge to humidifier water, all family members showed precipitin reactions |
| 1 female | Hypersensitivity pneumonitis | Cephalosporium in home humidifier | Precipitins to antigens |
| 2 asthmatics | Asthmatic episodes | Yeast contaminated aerosols in humidifier | Recurrent symptoms on re-exposure |
| 1 female | Pneumonitis, recurrent chills fever, cough, dyspnea | Thermophilic actinomycetes in home humidifier | Positive bronchial challenge |
| 1 male | Hypersensitivity pneumonitis | Thermoactinomyces vulgaris in console home humidifier | Symptoms disappeared when humidifier removed, precipitating antibodies |
| 1 female | Hypersensitivity pneumonitis | Thermoactinomyces vulgaris in humidifier | Precipitins against agent |

From Arundel et al. (1986).

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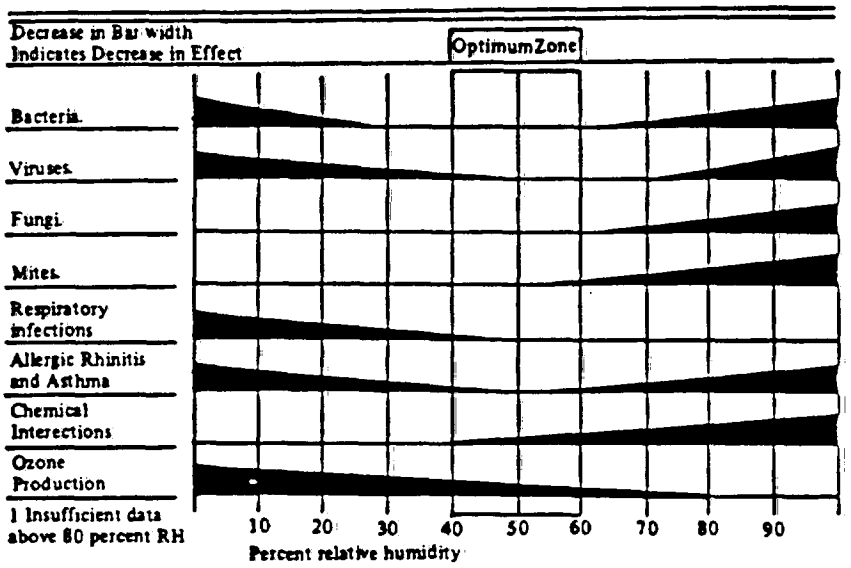
Figure 3. Optimum relative humidity range for minimizing adverse health effects.




From Arundel et al. (1986).

the humidity and growth of various types of bacteria, viruses, fungi, and mites, respiratory infections, atopic reactions, chemical interactions, and ozone production. As one can see from this figure, the growth of organic materials is minimized if the relative humidity in a building is maintained between 40% and 60%.

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## CONCLUSION

Modern office and public buildings create an indoor environment that is poorly understood and may be exceedingly hostile to occupants unless its hazards are clearly recognized. Because of the multiplicity of hazards and the chain of events that lead to physiological reactions in humans to contaminated indoor environments, no single and/or simple solution(s) is available. Adequate attention to ventilation and fresh air supply will eliminate or minimize many of the eye, throat, nose, and/or skin irritations caused by chemicals in the air by diluting them to a safe level. However, insofar as outdoor air contains a large number of toxic pollutants, it should be clear that a well ventilated building may only be able to maintain outdoor levels of cleanliness.

There are some instances, however, in which modern technology can improve and maintain the cleanliness of indoor air as compared to outdoor air. For instance, adequate filtering systems may remove much of the dust found outdoors and air conditioning and heating systems can adjust for climatic conditions. These technologies, however, need to be applied with a full understanding of the possible effect that application might have on human health. For example, while filters will remove unwanted dusts and respirable particles from the outdoor air, such filters also offer a medium for growth for a variety of bacteria and other infectious agents. Therefore, filters should be changed very frequently, which is a building maintenance expense. Similarly, aerosols from cooling towers need to be kept away from fresh air intakes in buildings. This requires careful planning in the placement of these air vents. (In addition, fresh air intake vents should not be placed near street level or garages where they will take in exhaust from motor vehicles and exhaust vents).

Finally a working relationship needs to be established between building engineers, architects, and epidemiologists. For instance, in studying a building with my associate, Elia Sterling (architect), we found that the high absentee rate and prevalence of irritation complaints was caused by the formation of indoor smog. A large amount of air was passed over high ultraviolet producing lighting fixtures. The impact of the high level of ultraviolet (*UV*)

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light on the air stream was similar to that of the sun on outdoor pollutants. Ultraviolet light is a cause of outdoor smog in such cities as San Francisco or Los Angeles. Similar UV emissions can cause the formation of smog indoors. We learned from this particular building that air vents should not be constructed to serve as lighting fixtures and high UV sunlight simulating fluorescent lighting should not be used (*Sterling, E. & Sterling, T.* 1983). We also learned that the epidemiology of sick buildings requires a multidisciplinary approach that should include architects, industrial hygienists and ventilation engineers.

## REFERENCES


APOL A.G. 1981. Health Hazard Evaluation Report, HETA 81-177, 1778-1988. University of Washington, Seattle, WA. NIOSH, Cincinnati, OH.

ARUNDEL A., STERLING E.M., BIGGIN J.H., STERLING T.D. 1986. Indirect Effects of Relative Humidity in Indoor Environments. Environmental Health Perspectives, 65:351-361.

BERGLUND B., LINDVALL T., SUNDELL T. (eds) 1984. Indoor Air (Vols. 1-5) Proceedings 3rd International Conference on Indoor Air Quality and Climate, Stockholm, August 20-23.

CHIO A., PIACITELLI L. 1985. Health Hazard Evaluation HETA 85- 196. Family Development Inc. Office, Parkersburg, WV. NIOSH, Cincinnati, OH.

COLLETT C.W., STERLING E.M., WEINKAM J.J., STEEVES J., MCINTYRE E.D. 1987. The Building Performance Database: An Analytical Tool for Indoor Air Quality Research. Proceedings Indoor Air '87.

CHROSTEK W. 1981. Health Hazard Evaluation Report HETA 81-251- 925, Skil Corporation, Philadelphia, Pensylvania. National Institute for Occupational Safety and Health, Cincinnati, OH.

CHROSTEK W. 1981. Health Hazard Evaluation Report HETA 81-084- 916, State College, Kutztown, Pensylvania. National Institute for Occupational Safety and Health, Cincinnati, OH.




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Source: https://www.industrydocuments.ucsf.edu/docs/zqyx0000

CONWILL D.E., WERNER S.B., DRITZ S.K., BISSETT M., COFFEY E., NYGAARD G., BRADFORD L., MORRISON F.R., KNIGHT M.W. 1982. Legionellosis--the 1980 A San Francisco outbreak. American Review of Respiratory Disease, 126:666-669.

COYE M.J., BELANGER P.L. 1982. Health Hazard Evaluation Report, HETA 82-176-1236. U.S. Customs Service, Patrol Division Office. Treasure Island, San Francisco, CA. NIOSH, Cincinnati, OH.

FANNICK N. 1980. Health Hazard Evaluation HE 80-6-667, Seiser and Wilpon, New York, NY. NIOSH, Cincinnati, OH.

FANNICK N. 1981. Health Hazard Evaluation Report HETA 81-241-970. National Park Service, Patchogue, NY. NIOSH, Cincinnati, OH.

FINNEGAN M.J., PICKERING C.A., BURGE P.S. 1984. The sick building syndrome: prevalence studies. British Medical Journal, 289:1573-1575.

FISHER-HOCH S.P., BARTLETT C.L., TOBIN J.O., GILLETT M.B., NELSON A.M., PRITCHARD J.E., SMITH M.G., SWANN R.A., TALBOT J.M., THOMAS J.A. 1981. Investigation and control of an outbreak of legionnaires' disease in a district general hospital. Lancet, 1:9932-9936.

FRASER D.W., TSAI T.R., ORENSTEIN W., PARKIN W.E., BEECHAM H.J., SHARRAR R.G., HARRIS J., MALLISON G.F., MARTIN S.M., MCDADE J.E., SHEPARD C.C., BRACHMAN P.S., Field Investigation Team. 1977. Legionnaires' Disease. Description of an Epidemic of Pneumonia. The New England Journal of Medicine, 297:1189-1197.

FRIEDMAN S., SPITALNY K., BARBAREE J., FAUR Y., MCKINNEY R. 1987. Pontiac Fever outbreak associated with a cooling tower. American Journal of Public Health, 77:568-572.

GARBE P.L., DAVIS B.J., WEISFELD J.S. 1985. Nosocomial Legionnaires' disease. Epidemiologic demonstration of cooling towers as a source. Journal of the American Medical Association, 254:521-524.

GRIST N.R., REID D., NAJERA R. 1979. Legionnaires' disease and the traveller. Annals of Internal Medicine, 90:563-564.

GUNTER B.J. 1981. Health Hazard Evaluation Report, HETA 81-057-905. Jefferson County Mental Health Center, Wheat Ridge, CO. NIOSH, Cincinnati, OH.



2023383856

Source: https://www.industrydocuments.ucsf.edu/docs/zqyx0000

GUNTER B.J. 1981. Health Hazard Evaluation Report, HETA 81-006- 849. Mountain Bell, Fort Collins, CO. NIOSH, Cincinnati, OH.

GUNTER B.J., THORBURN T.W. 1981. Health Hazard Evaluation Report HETA 81-108-883. Tri Valley Federal Credit Union, East Helena, Montana. NIOSH, Cincinnati, OH.

HAWKINS L.H. 1985. "Sick Building Syndrome" - Possible causes and symptoms. Paper presented at The Institute of Occupational Safety and Health Seminar on Office Safety, June 28.

HEDGE A. 1984a. Suggestive evidence for a relationship between office design and self-reports of ill health amongst office workers in the United Kingdom. Journal of Architectural and Planning Research, 1:163-174.

HEDGE A. 1984b. Ill health among office workers: an examination of the relationship between office design and employees well- being. In: E. Granjean (ed.) Ergonomics and Health in Modern Offices, page 46-51, London: Taylor & Francis.

HEDGE A., STERLING E.M., STERLING T.D. 1986. Building Illness indices based on questionnaire responses. Proceedings IAQ '86 Managing Indoor Air for Health and Energy Conservation.

KAUFMANN A.F., MCDADE J.E., PATTON C.M., BENNETT J.V., SKALIY P., FEELEY J.C., ANDERSON D.C., POTTER M.E., NEWHOUSE V.F., GREGG M.B., BRACHMAN P.S. 1981. Pontiac fever: isolation of the etiologic agent (Legionella pneumophillia) and demonstration of its mode of transmission. American Journal of Epidemiology, 114:337-347.

KIRKBRIDE J. 1985. Sick Building Syndrome: Causes and Effects. Health and Welfare Canada, Ottawa, Ontario.

KONOPINSKI V.J. 1980. Formaldehyde in Office and Commercial Environments. Indiana State Board of Health, Indianapolis, Indiana.

KREISS K. 1981. Pueblo Fiberglass Investigation. Colorado Department of Health, Division of Disease Control and Epidemiology.

KREISS K., GONZALEZ M., CONRIGHT K., SCHEERE A. 1981. Respiratory Irritation due to Carpet Shampoo: Two Outbreaks. Centers for Disease Control, Atlanta, GA.

MCMANUS K.P. 1985. Health Hazard Evaluation HETA 85-396. Barnes Building, Boston, MA. NIOSH (National Institute for Occupational Safety and Health), Cincinnati, OH.



2023383857

Source: https://www.industrydocuments.ucsf.edu/docs/zqyx0000

MELIUS J., WALLINGFORD K., KEENLYSIDE R., CARPENTER J. 1984. Indoor Air Quality - The NIOSH Experience. Annual American Conference of Government Industrial Hygienists.

NUDELMAN H. 1979. SP 78-2, St. Luke's Hospital, New York, NY. Interdepartmental Memo, City of New York.

PRYOR P., RENO S.J. 1981. Health Hazard Evaluation HETA 81-305- 961. Aurora Schools, Aurora, CO. NIOSH, Cincinnati, OH.

ROBERTSON A.S., BURGE P.S., HEDGE A., SIMMS J., GILL F.S., FINNEGAN M.J., PICKERING C.A., DALTON G. 1985. Comparison of health problems related to work and environmental measurements in two office buildings with different ventilation systems. British Medical Journal, 291:373-376.

ROBINSON P.A., WINKLER W.G., GREGG M.B. 1980. EPI 80-41-2. Respiratory Illness in Conference Participants - Washington, D.C. Public Health Service, CDC, Atlanta, GA.

SALISBURY S. 1981. Health Hazard Evaluation Report, HETA 81-002- 875. Atlanta Jewish Federation, GA. NIOSH, Cincinnati, OH.

SHOEMAKER W. 1977. Health Hazard Evaluation 76-70-367, Sperry Univac Corporation, Philadelphia, Pensylvania. National Institute for Occupational Safety and Health, Cincinnati, OH.

STERLING E., STERLING T. 1983. The impact of different ventilation levels and fluorescent lighting types on building illness: An experimental study. Canadian Journal of Public Health, 74:385-392.

STOLWIJK J.A.J., PIERCE J.B. 1984. The "sick building syndrome. In: Berlund B., Lindvall T., Sundell J. (eds.) Indoor Air, Vol. 1: Recent advances in the health sciences and technology, Proceedings of the International Conference on Indoor Air Quality and Climate, Stockholm, August 20-24, pp. 23-29.

THOBURN T.W. 1981. Health Hazard Evaluation Report, HETA 81-005- 834. Public Employees Retirement Association. Denver, Colorado. NIOSH, Cincinnati, OH.

TURIEL I., HOLLOWELL C.D., MIKSCH R.R., RUDY J.V., YOUNG R.A. 1983. The effects of reduced ventilation on indoor air quality in an office building. Atmospheric Environment, 17:51-64.



2023383858

Source: https://www.industrydocuments.ucsf.edu/docs/zqyx0000

WORLD HEALTH ORGANIZATION (WHO), 1982. Indoor Air Pollutants: Exposure and Health Effects Assessment. (Working Group Report, Nordlingen, Euro reports and studies no. 78, WHO, Copenhagen).

WORLD HEALTH ORGANIZATION (WHO), 1983. Indoor air pollutants: exposure and health effects, EURO Reports and Studies 78.

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ASHRAE 62-1989

# ASHRAE™ STANDARD

## Ventilation for Acceptable Indoor Air Quality

Approved by the ASHRAE Standards Committee March 1, 1989; approved by the Board of Directors June 29, 1989.

ASHRAE Standards are updated on a five-year cycle; the date following the Standard number is the year of approval. The latest copies may be purchased from the ASHRAE Publications Sales Department, 1791 Tullie Circle, NE, Atlanta, GA 30329.

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**This Foreword is not part of this Standard but is included for information purposes only.**

## FOREWORD

ASHRAE's first ventilation standard was ASHRAE Standard 62-73, "Standard for Natural and Mechanical Ventilation" (see Ref i). The standard provided a prescriptive approach to ventilation by specifying both minimum and recommended outdoor air flow rates to obtain acceptable indoor air quality for a variety of indoor spaces. This standard is still referenced in many building codes and was referenced by ASHRAE's first energy Standard, 90-75 (see Ref ii), which specified use of the minimum outdoor air flow rates from 62-73. The revised energy standard, 90A-1980 (see Ref iii), also took this approach.

Under the normal five-year review cycle, the multidisciplinary standards project committee appointed in 1978 addressed the question of whether these minimum values could be defended under all conditions. The revised Standard 62-1981, "Ventilation for Acceptable Indoor Air Quality" (see Ref. iv), recommended outdoor air flow rates for smoking-permitted and for smoking-prohibited conditions in most spaces. The 1981 standard also introduced an alternative air quality procedure to permit innovative, energy-conserving ventilation practices. The alternative procedure allowed the engineer to use whatever amount of outdoor air he deemed necessary if he could show that the levels of indoor air contaminants were held below recommended limits. However, some of the users of Standard 62-1981 found the application of the different ventilation rates for smoking and nonsmoking areas confusing, and the recommended maximum concentration of formaldehyde was challenged. For these reasons and in the light of rapidly changing technology, ASHRAE authorized an early review of Standard 62-1981 beginning in January 1983.

This revised Standard retains the two procedures for ventilation design, the Ventilation Rate Procedure and the Air Quality Procedure. The goals of achieving acceptable indoor air quality and of minimizing energy consumption appear to imply a compromise. An interdisciplinary committee of engineers, architects, chemists, physiologists, product manufacturers, and industry representatives has endeavored to achieve the necessary balance between energy consumption and indoor air quality in this Standard. It must be recognized, however, that the conditions specified by this Standard must be achieved during the operation of buildings as well as in the design of the buildings if acceptable indoor air quality is to be achieved. To facilitate this, the Standard includes requirements for ventilation design documentation to be provided for system operation. The purpose of the Standard is to specify minimum ventilation rates and indoor air quality that will be acceptable to human occupants and are intended to avoid adverse health effects. For substantive information on health effects, the Standard must rely on recognized authorities and their specific recommendations. Therefore, with respect to tobacco smoke and other contaminants, this Standard does not, and cannot, ensure the avoidance of all possible adverse health effects, but it reflects recognized consensus criteria and guidance.

The Appendices are not part of this standard but are included for information purposes only.

Upon publication of this revised standard, an addendum process will be initiated to update references to other standards and government publications.

### References


i. *ASHRAE Standard 62-73 (ANSI B 194.1-1977), Standards for Natural and Mechanical Ventilation*. American Society of Heating, Refrigerating, and Air-Conditioning Engineers, Inc., Atlanta, GA. 1977.

ii. *ASHRAE Standard 90-75, Energy Conservation in New Building Design*. American Society of Heating, Refrigerating, and Air-Conditioning Engineers, Inc., Atlanta, GA. 1975.

iii. *ANSI/ASHRAE/IES Standard 90A-1980, Energy Conservation in New Building Design*. American Society of Heating, Refrigerating, and Air-Conditioning Engineers, Inc., Atlanta, GA. 1980.

iv. *ASHRAE Standard 62-1981, Ventilation for Acceptable Indoor Air Quality*. American Society of Heating, Refrigerating, and Air-Conditioning Engineers, Inc., Atlanta, GA. 1981.


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## 1. PURPOSE

To specify minimum ventilation rates and indoor air quality that will be acceptable to human occupants and are intended to avoid adverse health effects.

## 2. SCOPE

This Standard applies to all indoor or enclosed spaces that people may occupy, except where other applicable standards and requirements dictate larger amounts of ventilation than this Standard. Release of moisture in residential kitchens and bathrooms, locker rooms, and swimming pools is included in the scope of this Standard.

## 3. DEFINITIONS (see Fig. 1)

**absorption:** the process of one substance entering into the inner structure of another.

**acceptable indoor air quality:** air in which there are no known contaminants at harmful concentrations as determined by cognizant authorities and with which a substantial majority (80% or more) of the people exposed do not express dissatisfaction.

**adsorption:** the adhesion of a thin film of liquid or gases to the surface of a solid substance.

**air-cleaning system:** a device or combination of devices applied to reduce the concentration of airborne contaminants, such as microorganisms, dusts, fumes, respirable particles, other particulate matter, gases, and/or vapors in air.

**air conditioning:** the process of treating air to meet the requirements of a conditioned space by controlling its temperature, humidity, cleanliness, and distribution.

**air, ambient:** the air surrounding an object.

**air, exhaust:** air removed from a space and not reused therein.

**air, makeup:** outdoor air supplied to replace exhaust air and exfiltration.

**air, outdoor:** air taken from the external atmosphere and, therefore, not previously circulated through the system.

**air, recirculated:** air removed from the conditioned space and intended for reuse as supply air.

**air, return:** air removed from a space to be then recirculated or exhausted.

**air, supply:** that air delivered to the conditioned space and used for ventilation, heating, cooling, humidification, or dehumidification.

**air, transfer:** the movement of indoor air from one space to another.

**air, ventilation:** that portion of supply air that is outdoor air plus any recirculated air that has been treated for the purpose of maintaining acceptable indoor air quality.

**chemisorb:** to take up and hold, usually irreversibly, by chemical forces.

**concentration:** the quantity of one constituent dispersed in a defined amount of another (see Appendix A).

**conditioned space:** that part of a building that is heated or cooled, or both, for the comfort of occupants.

**contaminant:** an unwanted airborne constituent that may reduce acceptability of the air.

**dust:** an air suspension of particles (aerosol) of any solid material, usually with particle size less than 100 micrometers ($\mu$m).

**energy recovery ventilation system:** a device or combination of devices applied to provide the outdoor air for ventilation in which energy is transferred between the intake and exhaust airstreams.

**exfiltration:** air leakage outward through cracks and interstices and through ceilings, floors, and walls of a space or building.

**fumes:** airborne particles, usually less than 1 micrometer in size, formed by condensation of vapors, sublimation, distillation, calcination, or chemical reaction.

**gas:** a state of matter in which substances exist in the form of nonaggregated molecules, and which, within acceptable limits of accuracy, satisfies the ideal gas laws; usually a highly superheated vapor.

**infiltration:** air leakage inward through cracks and interstices and through ceilings, floors, and walls of a space or building.

**microorganism:** a microscopic organism, especially a bacterium, fungus, or a protozoan.

**natural ventilation:** the movement of outdoor air into a space through intentionally provided openings, such as windows and doors, or through nonpowered ventilators or by infiltration.

**occupied zone:** the region within an occupied space between planes 3 and 72 in. (75 and 1800 mm) above the floor and more than 2 ft (600 mm) from the walls or fixed air-conditioning equipment (see ASHRAE Standard 55-1981, Ref.1)

**odor:** a quality of gases, liquids, or particles that stimulates the olfactory organ.

**oxidation:** a reaction in which oxygen combines with another substance.

**particulate matter:** a state of matter in which solid or liquid substances exist in the form of aggregated molecules or particles. Airborne particulate matter is typically in the size range of 0.01 to 100 micrometers.

**plug flow:** a flow regime where the flow is predominately in one direction and contaminants are swept along with the flow.

**smoke:** the airborne solid and liquid particles and gases that evolve when a material undergoes pyrolysis or combustion. Note: chemical smoke is excluded from this definition.

**total suspended particulate matter:** the mass of particles suspended in a unit of volume of air when collected by a high-volume air sampler.

**respirable particles:** respirable particles are those that penetrate into and are deposited in the nonciliated portion

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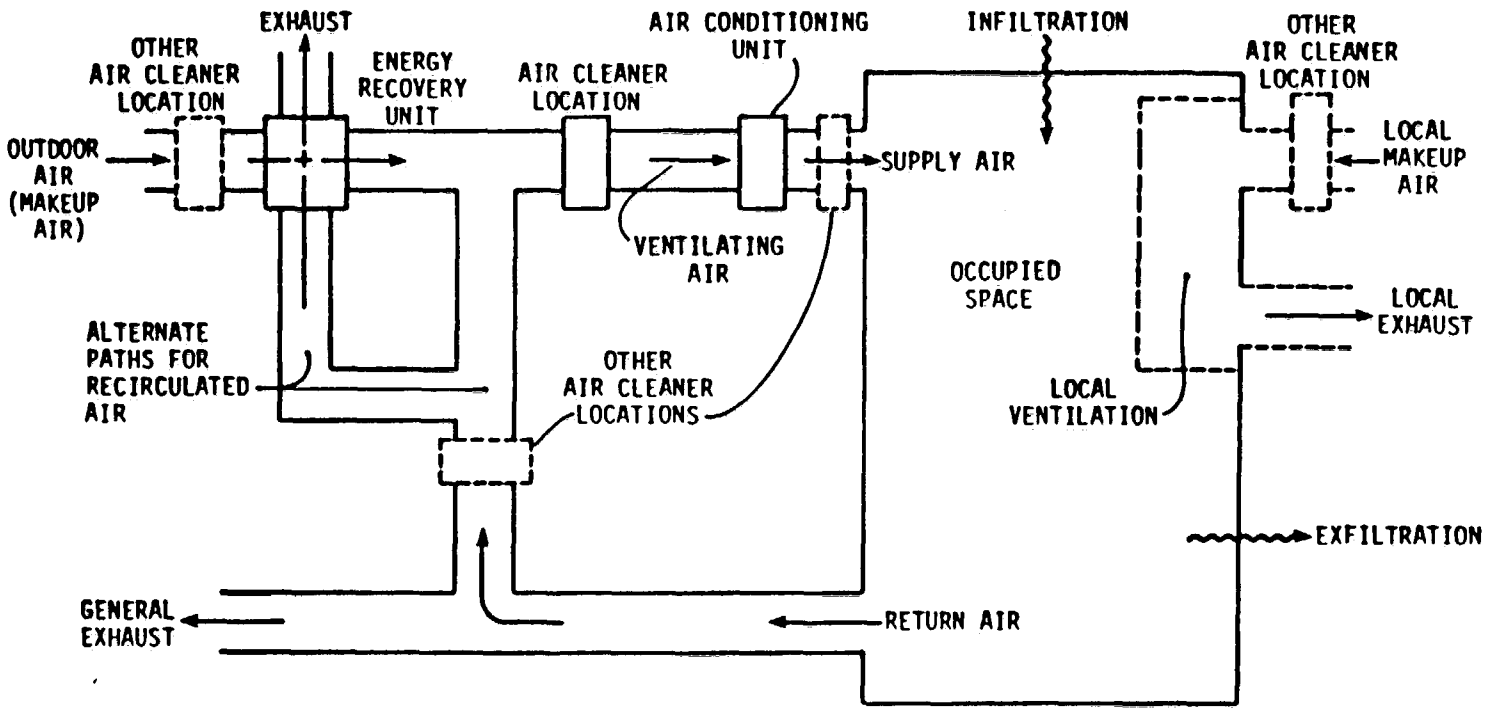


*Fig. 1 Ventilation System*



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of the lung. Particles greater than 10 micrometers aerodynamic diameter are not respirable.

**vapor:** a substance in gas form, particularly one near equilibrium with its condensed phase, which does not obey the ideal gas laws; in general, any gas below its critical temperature.

**ventilation:** the process of supplying and removing air by natural or mechanical means to and from any space. Such air may or may not be conditioned.

## 4. CLASSIFICATION

This Standard specifies alternative procedures to obtain acceptable air quality indoors:

**4.1 Ventilation Rate Procedure:** Acceptable air quality is achieved by providing ventilation air of the specified quality and quantity to the space (see 6.1). or

**4.2 Indoor Air Quality Procedure:** Acceptable air quality is achieved within the space by controlling known and specifiable contaminants (see 6.2).

Whenever the Ventilation Rate Procedure is used, the design documentation should clearly state that this method was used and that the design will need to be re-evaluated if, at a later time, space use changes occur or if unusual contaminants or unusually strong sources of specific contaminants are to be introduced into the space. If such conditions are known at the time of the original design, the use of the Indoor Air Quality Procedure may be indicated.

The Indoor Air Quality Procedure could result in a ventilation rate lower than would result from the first procedure, but the presence of a particular source of contamination in the space may result in increased ventilation requirements. Change in space use, contaminants, or operation may require a re-evaluation of the design and implementation of needed changes.

## 5. SYSTEMS AND EQUIPMENT

**5.1** Ventilating systems may be mechanical or natural. When mechanical ventilation is used, provision for air flow measurement should be included. When natural ventilation and infiltration are relied upon, sufficient ventilation shall be demonstrable. When infiltration and natural ventilation are insufficient to meet ventilation air requirements, mechanical ventilation shall be provided. The use of energy recovery ventilation systems should be considered for energy conservation purposes in meeting ventilation requirements.

**5.2** Ventilating systems shall be designed and installed so that the ventilation air is supplied throughout the occupied zone. The design documentation shall state assumptions that were made in the design with respect to ventilation rates and air distribution.

**5.3** Ventilating systems shall be designed and installed so that they do not cause conditions that conflict with ASHRAE Standard 55-1981, "Thermal Environmental Conditions for Human Occupancy" (Ref. 1).

**5.4** When the supply of air is reduced during times the space is occupied (e.g., in variable-air-volume systems), provision shall be made to maintain acceptable indoor air quality throughout the occupied zone.

**5.5** Ventilating systems should be designed to prevent reentrainment of exhaust contaminants, condensation or freeze-ups (or both), and growth of microorganisms. Makeup air inlets and exhaust air outlets shall be located to avoid contamination of the makeup air. Contaminants from sources such as cooling towers, sanitary vents, vehicular exhaust from parking garages, loading docks, and street traffic should be avoided. This is a special problem in buildings where stack effect draws contaminants from these areas into the occupant space. Where soils contain high concentrations of radon, ventilation practices that place crawlspaces, basements, or underground ductwork below atmospheric pressure will tend to increase radon concentrations in buildings and should be avoided (see Appendix C).

**5.6** Ventilating ducts and plenums shall be constructed and maintained to minimize the opportunity for growth and dissemination of microorganisms through the ventilation system. Construction also shall comply with applicable standards such as NFPA 90A, 90B, and SMACNA (Refs. 2-6).

**5.7** Contaminants from stationary local sources within the space shall be controlled by collection and removal as close to the source as practicable. (See Ref 7, "Industrial Ventilation—Manual of Recommended Practice")

**5.8** Fuel-burning appliances, including fireplaces located indoors, shall be provided with sufficient air for combustion and adequate removal of combustion products. When infiltration supplies all or part of the combustion air, the supply rate of air shall be demonstrable (Appendix B shows one method of demonstrating adequate combustion air). The operation of clothes dryers and exhaust fans may require introduction of additional makeup air to avoid interference with fuel-burning appliances. Combustion system, kitchen, bathroom, and clothes dryer vents shall not be exhausted into attics, crawlspaces, or basements.

**5.9** Airborne particulate contaminants vary in size, as shown in Fig. 2. Microorganisms, dusts, fumes, smoke, and other particulate matter may be captured by air filters. Many bacteria (99% exceed 1 micrometer in size) are attached to larger particles such as human skin flakes. Viruses generally occur in clusters or in and on other particles. Lung-damaging particles that may be retained in the lungs are 0.2 to 5 micrometers in size (see Fig. 2). When it is necessary to remove particulate contaminants, air filters or dust collectors should be used. Dust collectors, not air filters, should be used where the dust loading equals or exceeds 10 mg/m$^3$ (4 grains/1000 ft$^3$). Air filters and dust collectors shall be selected for the particle size and loading encountered. Filters shall be tested in accordance with ASHRAE Standard 52-76 (Ref 8) or MIL Std 282 (Ref 9). Dust collectors may be wet, dry, or electrostatic as required by particle size and loading (see Table 1, Chapter 11, *ASHRAE Handbook—1983 Equipment Volume* (Ref 10).

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**5.10** When compliance with this section does not provide adequate control of gaseous contaminants, methods based on sorption with or without oxidation or other scientifically proven technology shall be used. Such methods may be tailored to deal with a specific contaminant. A commonly used sorbent is activated carbon. The selection of gaseous contaminant control equipment for recirculation systems must consider the concentration, toxicity, annoyance, and odor properties of the contaminants present and the levels to which these must be reduced to be effective in maintaining air quality. The performance of gaseous contaminant removal devices often depends strongly on the physical and chemical properties of the individual contaminants present, on the temperature and humidity of the air, on the air velocity through the device, and its loading capacity.

**5.11** High humidities can support the growth of pathogenic or allergenic organisms (see Ref 20). Examples include certain species of fungi, associated mycotoxins, and dust mites. This growth is enhanced by the presence of materials with high cellulose, even with low nitrogen content, such as fiberboard, dust, lint, skin particles, and dander. Areas of concern include bathrooms and bedrooms. Therefore, bathrooms shall conform to the ventilation rates in Table 2.3. Relative humidity in habitable spaces preferably should be maintained between 30% and 60% relative humidity (see Ref 11) to minimize growth of allergenic or pathogenic organisms.

**5.12** Microbial contamination in buildings is often a function of moisture incursion from sources such as stagnant water in HVAC air distribution systems and cooling towers. Air-handling unit condensate pans shall be designed for self-drainage to preclude the buildup of microbial slime. Provision shall be made for periodic in-situ cleaning of cooling coils and condensate pans. Air-handling and fan coil units shall be easily accessible for inspection and preventive maintenance. Steam is preferred as a moisture source for humidifiers, but care should be exercised to avoid contamination from boiler water or steam supply additives. If cold water humidifiers are specified, the water shall originate from a potable source, and, if recirculated, the system will require frequent maintenance and blow-down. Care should be exercised to avoid particulate contamination due to evaporation of spray water. Standing water used in conjunction with water sprays in HVAC air distribution systems should be treated to avoid microbial buildup. If the

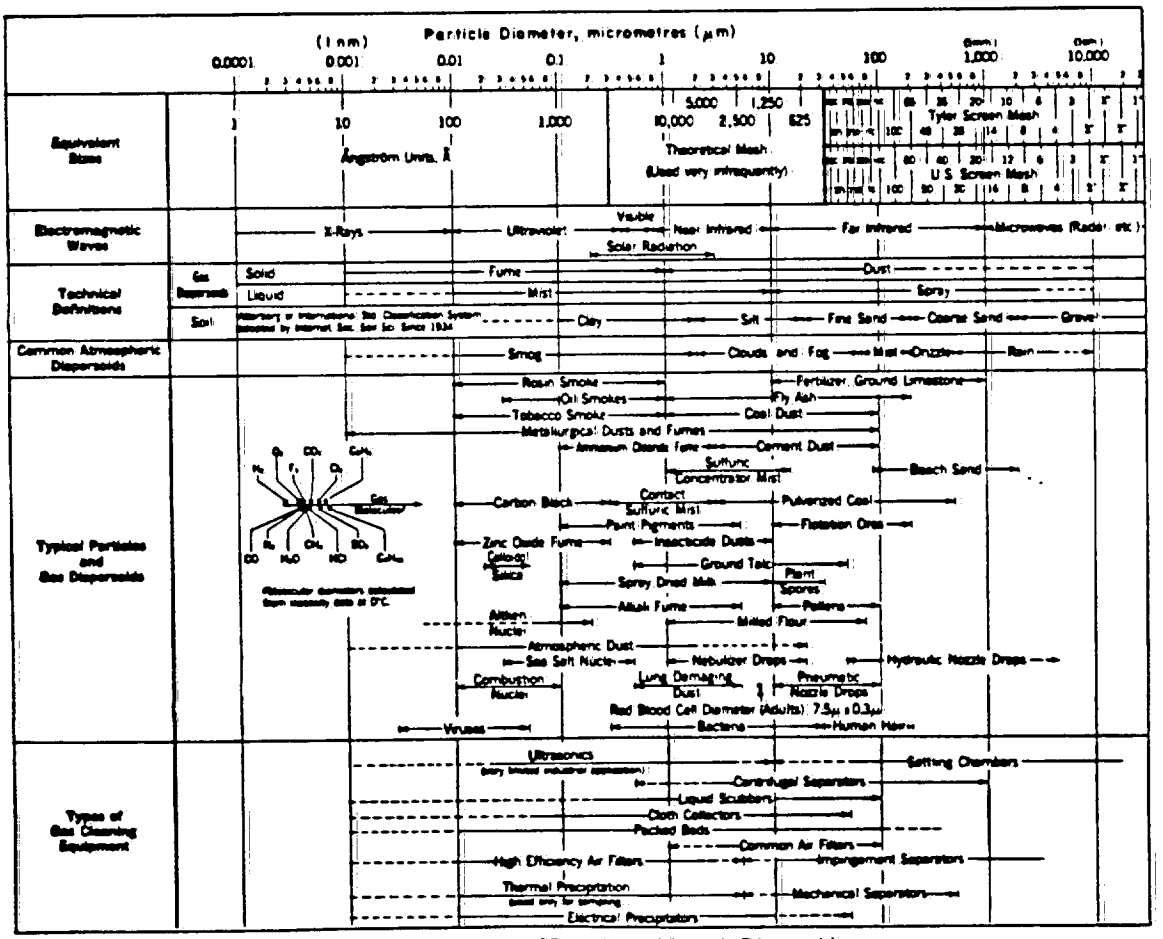


*Fig. 2 Characteristics of Particles and Particle Dispersoids*

*Courtesy of Stanford Research Institute.*

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relative humidity in occupied spaces and low velocity ducts and plenums exceeds 70%, fungal contamination (for example, mold, mildew, etc.) can occur. Special care should be taken to avoid entrainment of moisture drift from cooling towers into the makeup air and building vents.

## 6. PROCEDURES

Indoor air quality is a function of many parameters including outdoor air quality, the design of enclosed spaces, the design of the ventilation system, the way this system is operated and maintained, and the presence of sources of contaminants and the strength of such sources. This Standard deals with the design of a ventilation system as it is affected by all these factors, so that an acceptable level of indoor air quality can be provided. Design documentation shall clearly state which assumptions were used in the design so that the limits of the system in removing contaminants can be evaluated by others before the system is operated in a different mode or before new sources are introduced into the space.

Indoor air should not contain contaminants that exceed concentrations known to impair health or cause discomfort to occupants. Such contaminants include various gases, vapors, microorganisms, smoke, and other particulate matter. These may be present in makeup air or be introduced from indoor activities, furnishings, building materials, surface coatings, and air-handling and air treatment components. Deleterious factors include toxicity, radioactivity, potential to induce infection or allergies, irritants, extreme thermal conditions, and objectionable odors.

The Ventilation Rate Procedure (6.1) provides one way to achieve acceptable air quality. This procedure prescribes the rate at which ventilation air must be delivered to a space and various means to condition that air. The ventilation rates in Table 2 are derived from physiological considerations, subjective evaluations, and professional judgments (see Refs 12-18).

The Indoor Air Quality Procedure (6.2) provides an alternative performance method for achieving acceptable air quality. This procedure uses one or more guidelines for the specification of acceptable concentrations of certain contaminants in indoor air but does not prescribe ventilation rates or air treatment methods.

**TABLE 1**
**National Primary Ambient-Air Quality Standards for Outdoor Air as Set by the U.S. Environmental Protection Agency (Ref 19)**

| Contaminant | Long term | | | Short term | | |
|---|---|---|---|---|---|---|
| | Concentration $\mu g/m^3$ | ppm | Averaging | Concentration $\mu g/m^3$ | ppm | Averaging |
| Sulfur dioxide | 80 | 0.03 | 1 year | 365 | 0.14 | 24 hours |
| Total Particulate | 75[a] | — | 1 year | 260 | — | 24 hours |
| Carbon monoxide | | | | 40,000 | 35 | 1 hour |
| Carbon monoxide | | | | 10,000 | 9 | 8 hours |
| Oxidants (ozone) | | | | 235[b] | 0.12[b] | 1 hour |
| Nitrogen dioxide | 100 | 0.055 | 1 year | | | |
| Lead | 1.5 | — | 3 months[c] | | | |

[a] Arithmetic mean

[b] Standard is attained when expected number of days per calendar year with maximal hourly average concentrations above 0.12 ppm (235 $\mu g/m^3$) is equal to or less than 1, as determined by Appendix H to subchapter C, 40 CFR 50

[c] Three-month period is a calendar quarter.

**6.1 Ventilation Rate Procedure:** This procedure prescribes:

- the outdoor air quality acceptable for ventilation
- outdoor air treatment when necessary
- ventilation rates for residential, commercial, institutional, vehicular, and industrial spaces
- criteria for reduction of outdoor air quantities when recirculated air is treated by contaminant-removal equipment
- criteria for variable ventilation when the air volume in the space can be used as a reservoir to dilute contaminants.

**6.1.1 Acceptable Outdoor Air.** This section describes a three-step procedure by which outdoor air shall be evaluated for acceptability:

**Step 1:** Contaminants in outdoor air do not exceed the concentrations listed in Table 1 as determined by one of the following conditions:

(a) Monitoring data of government pollution-control agencies, such as the U.S. Environmental Protection Agency (EPA) or equivalent state or local environmental protection authorities, show that the air quality of the area in which the ventilating system is located meets the requirements of Table 1. Conformity of local air to these standards may be determined by reference to the records of local authorities or of the National Aerometric Data Bank, Office of Air Quality Planning and Standards, EPA, Research Triangle Park, NC 27711, or

(b) The ventilating system is located in a community similar in population, geographic and meteorological settings, and industrial pattern to a community having acceptable air quality as determined by authorities having jurisdiction, or

(c) The ventilating system is located in a community with a population of less than 20,000 people, and the air is not influenced by one or more sources that cause substantial contamination, or

(d) Air monitoring for three consecutive months, as required for inclusion in the National Aerometric Data Bank, shows that the air quality meets or exceeds the requirements of Table 1 (as specified in Ref 19).

**Step 2:** If the outdoor air is thought to contain any contaminants not listed in Table 1, guidance on acceptable concentration levels may be obtained by reference to Appendix C.

Outdoor air requirements for ventilation of industrial building occupancies not listed in Table 2 may be determined by procedures presented in *1986 Industrial Ventilation—A Manual of Recommended Practice*, 1986 ed., published by the American Conference of Governmental Industrial Hygienists (ACGIH) (Ref 7).

**Step 3:** If after completing steps 1 and 2 there is still a reasonable expectation that the air is unacceptable, sampling shall be conducted in accordance with NIOSH pro-

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## TABLE 2
## OUTDOOR AIR REQUIREMENTS FOR VENTILATION*

### 2.1 COMMERCIAL FACILITIES (offices, stores, shops, hotels, sports facilities)

| Application | Estimated Maximum** Occupancy P/1000 ft² or 100 m² | Outdoor Air Requirements cfm/person | L/s·person | cfm/ft² | L/s·m² | Comments |
|---|---|---|---|---|---|---|
| **Dry Cleaners, Laundries** | | | | | | Dry-cleaning processes may require more air. |
| Commercial laundry | 10 | 25 | 13 | | | |
| Commercial dry cleaner | 30 | 30 | 15 | | | |
| Storage, pick up | 30 | 35 | 18 | | | |
| Coin-operated laundries | 20 | 15 | 8 | | | |
| Coin-operated dry cleaner | 20 | 15 | 8 | | | |
| **Food and Beverage Service** | | | | | | |
| Dining rooms | 70 | 20 | 10 | | | |
| Cafeteria, fast food | 100 | 20 | 10 | | | |
| Bars, cocktail lounges | 100 | 30 | 15 | | | Supplementary smoke-removal equipment may be required. |
| Kitchens (cooking) | 20 | 15 | 8 | | | Makeup air for hood exhaust may require more ventilating air. The sum of the outdoor air and transfer air of acceptable quality from adjacent spaces shall be sufficient to provide an exhaust rate of not less than 1.5 cfm/ft² (7.5 L/s·m²). |
| **Garages, Repair, Service Stations** | | | | | | |
| Enclosed parking garage | | | | 1.50 | 7.5 | Distribution among people must consider worker location and concentration of running engines; stands where engines are run must incorporate systems for positive engine exhaust withdrawal. Contaminant sensors may be used to control ventilation. |
| Auto repair rooms | | | | 1.50 | 7.5 | |
| **Hotels, Motels, Resorts, Dormitories** | | | | cfm/room | L/s·room | Independent of room size. |
| Bedrooms | | | | 30 | 15 | |
| Living rooms | | | | 30 | 15 | |
| Baths | | | | 35 | 18 | Installed capacity for intermittent use. |
| Lobbies | 30 | 15 | 8 | | | |
| Conference rooms | 50 | 20 | 10 | | | |
| Assembly rooms | 120 | 15 | 8 | | | |
| Dormitory sleeping areas | 20 | 15 | 8 | | | See also food and beverage services, merchandising, barber and beauty shops, garages. |
| Gambling casinos | 120 | 30 | 15 | | | Supplementary smoke-removal equipment may be required. |
| **Offices** | | | | | | |
| Office space | 7 | 20 | 10 | | | Some office equipment may require local exhaust. |
| Reception areas | 60 | 15 | 8 | | | |
| Telecommunication centers and data entry areas | 60 | 20 | 10 | | | |
| Conference rooms | 50 | 20 | 10 | | | Supplementary smoke-removal equipment may be required. |
| **Public Spaces** | | | | cfm/ft² | L/s·m² | |
| Corridors and utilities | | | | 0.05 | 0.25 | |
| Public restrooms, cfm/wc or urinal | | 50 | 25 | | | Mechanical exhaust with no recirculation is recommended. |
| Locker and dressing rooms | | | | 0.5 | 2.5 | |
| Smoking lounge | 70 | 60 | 30 | | | Normally supplied by transfer air, local mechanical exhaust; with no recirculation recommended. |
| **Elevators** | | | | 1.00 | 5.0 | Normally supplied by transfer air. |

* Table 2 prescribes supply rates of acceptable outdoor air required for acceptable indoor air quality. These values have been chosen to control $CO_2$ and other contaminants with an adequate margin of safety and to account for health variations among people, varied activity levels, and a moderate amount of smoking. Rational of $CO_2$ control is presented in Appendix D.

**Net occupiable space.

cedures (see Refs 21 and 22). Local and national aerometric data banks may contain information on some unregulated pollutants. Finally, acceptable outdoor air quality should be evaluated using the definition for acceptable indoor air quality in Section 3.



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TABLE 2
OUTDOOR AIR REQUIREMENTS FOR VENTILATION* (*Continued*)
2.1 COMMERCIAL FACILITIES (offices, stores, shops, hotels, sports facilities)

| Application | Estimated Maximum** Occupancy P/1000 ft² or 100 m² | Outdoor Air Requirements cfm/person | Outdoor Air Requirements L/s·person | Outdoor Air Requirements cfm/ft² | Outdoor Air Requirements L/s·m² | Comments |
|---|---|---|---|---|---|---|
| **Retail Stores, Sales Floors, and Show Room Floors** | | | | | | |
| Basement and street | 30 | | | 0.30 | 1.50 | |
| Upper floors | 20 | | | 0.20 | 1.00 | |
| Storage rooms | 15 | | | 0.15 | 0.75 | |
| Dressing rooms | | | | 0.20 | 1.00 | |
| Malls and arcades | 20 | | | 0.20 | 1.00 | |
| Shipping and receiving | 10 | | | 0.15 | 0.75 | |
| Warehouses | 5 | | | 0.05 | 0.25 | |
| Smoking lounge | 70 | 60 | 30 | | | Normally supplied by transfer air, local mechanical exhaust; exhaust with no recirculation recommended. |
| **Specialty Shops** | | | | | | |
| Barber | 25 | 15 | 8 | | | |
| Beauty | 25 | 25 | 13 | | | |
| Reducing salons | 20 | 15 | 8 | | | |
| Florists | 8 | 15 | 8 | | | Ventilation to optimize plant growth may dictate requirements. |
| Clothiers, furniture | | | | 0.30 | 1.50 | |
| Hardware, drugs, fabric | 8 | 15 | 8 | | | |
| Supermarkets | 8 | 15 | 8 | | | |
| Pet shops | | | | 1.00 | 5.00 | |
| **Sports and Amusement** | | | | | | |
| Spectator areas | 150 | 15 | 8 | | | When internal combustion engines are operated for maintenance of playing surfaces, increased ventilation rates may be required. |
| Game rooms | 70 | 25 | 13 | | | |
| Ice arenas (playing areas) | | | | 0.50 | 2.50 | |
| Swimming pools (pool and deck area) | | | | 0.50 | 2.50 | Higher values may be required for humidity control. |
| Playing floors (gymnasium) | 30 | 20 | 10 | | | |
| Ballrooms and discos | 100 | 25 | 13 | | | |
| Bowling alleys (seating areas) | 70 | 25 | 13 | | | |
| **Theaters** | | | | | | Special ventilation will be needed to eliminate special stage effects (e.g., dry ice vapors, mists, etc.). |
| Ticket booths | 60 | 20 | 10 | | | |
| Lobbies | 150 | 20 | 10 | | | |
| Auditorium | 150 | 15 | 8 | | | |
| Stages, studios | 70 | 15 | 8 | | | |
| **Transportation** | | | | | | Ventilation within vehicles may require special considerations. |
| Waiting rooms | 100 | 15 | 8 | | | |
| Platforms | 100 | 15 | 8 | | | |
| Vehicles | 150 | 15 | 8 | | | |
| **Workrooms** | | | | | | |
| Meat processing | 10 | 15 | 8 | | | Spaces maintained at low temperatures (−10 °F to + 50 °F, or −23 °C to + 10 °C) are not covered by these requirements unless the occupancy is continuous. Ventilation from adjoining spaces is permissible. When the occupancy is intermittent, infiltration will normally exceed the ventilation requirement. (See Ref 18). |

* Table 2 prescribes supply rates of acceptable outdoor air required for acceptable indoor air quality. These values have been chosen to control $CO_2$ and other contaminants with an adequate margin of safety and to account for health variations among people, varied activity levels, and a moderate amount of smoking. Rational of $CO_2$ control is presented in Appendix D.
**Net occupiable space.

6.1.2 **Outdoor Air Treatment.** If the outdoor air contaminant levels exceed the values given in 6.1.1 (Table 1), the air should be treated to control the offending contaminants. Air-cleaning systems suitable for the particle size encountered should be used. For removal of gases and vapors, appropriate air-cleaning systems should be used. Where the best available, demonstrated, and proven technology does not allow for the removal of contaminants, the amount of outdoor air may be reduced during periods of high contaminant levels, such as those generated by rush-hour



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TABLE 2
OUTDOOR AIR REQUIREMENTS FOR VENTILATION* (*Concluded*)

2.1 COMMERCIAL FACILITIES (offices, stores, shops, hotels, sports facilities)

| Application | Estimated Maximum** Occupancy P/1000 ft² or 100 m² | Outdoor Air Requirements cfm/person | Outdoor Air Requirements L/s·person | Outdoor Air Requirements cfm/ft² | Outdoor Air Requirements L/s·m² | Comments |
|---|---|---|---|---|---|---|
| Photo studios | 10 | 15 | 8 | | | |
| Darkrooms | 10 | | | 0.50 | 2.50 | |
| Pharmacy | 20 | 15 | 8 | | | |
| Bank vaults | 5 | 15 | 8 | | | |
| Duplicating, printing | | | | 0.50 | 2.50 | Installed equipment must incorporate positive exhaust and control (as required) of undesirable contaminants (toxic or otherwise). |
| **2.2 INSTITUTIONAL FACILITIES** | | | | | | |
| **Education** | | | | | | |
| Classroom | 50 | 15 | 8 | | | |
| Laboratories | 30 | 20 | 10 | | | Special contaminant control systems may be required for processes or functions including laboratory animal occupancy. |
| Training shop | 30 | 20 | 10 | | | |
| Music rooms | 50 | 15 | 8 | | | |
| Libraries | 20 | 15 | 8 | | | |
| Locker rooms | | | | 0.50 | 2.50 | |
| Corridors | | | | 0.10 | 0.50 | |
| Auditoriums | 150 | 15 | 8 | | | |
| Smoking lounges | 70 | 60 | 30 | | | Normally supplied by transfer air. Local mechanical exhaust with no recirculation recommended. |
| **Hospitals, Nursing and Convalescent Homes** | | | | | | |
| Patient rooms | 10 | 25 | 13 | | | Special requirements or codes and pressure relationships may determine minimum ventilation rates and filter efficiency. Procedures generating contaminants may require higher rates. |
| Medical procedure | 20 | 15 | 8 | | | |
| Operating rooms | 20 | 30 | 15 | | | |
| Recovery and ICU | 20 | 15 | 8 | | | |
| Autopsy rooms | | | | 0.50 | 2.50 | Air shall not be recirculated into other spaces. |
| Physical Therapy | 20 | 15 | 8 | | | |
| **Correctional Facilities** | | | | | | |
| Cells | 20 | 20 | 10 | | | |
| Dining halls | 100 | 15 | 8 | | | |
| Guard stations | 40 | 15 | 8 | | | |

* Table 2 prescribes supply rates of acceptable outdoor air required for acceptable indoor air quality. These values have been chosen to control $CO_2$ and other contaminants with an adequate margin of safety and to account for health variations among people, varied activity levels, and a moderate amount of smoking. Rationale for $CO_2$ control is presented in Appendix D.

** Net occupiable space.

traffic. The need to control offending contaminants may depend on local regulations that require specific control measures.

**6.1.3 Ventilation Requirements.** Indoor air quality shall be considered acceptable if the required rates of acceptable outdoor air in Table 2 are provided for the occupied space.

**Exceptions:**

1. Where unusual indoor contaminants or sources are present or anticipated, they shall be controlled at the source or the procedure of 6.2 shall be followed.

2. For those areas within industrial facilities not covered by Table 2, refer to TLVs—*Threshold Limit Values and Biological Exposure Indices for 1986-87*, American Conference of Governmental Industrial Hygienists (Ref 23).

Table 2 lists the required ventilation rates in cfm (L/s) per person or cfm/ft² (L/s·m²) for a variety of indoor spaces. In most cases, the contamination produced is presumed to be in proportion to the number of persons in the space. In other cases, the contamination is presumed to be chiefly due to other factors and the ventilating rates given are based on more appropriate parameters. Where appropriate, the table lists the estimated density of people for design purposes.

Where occupant density differs from that in Table 2, use the per occupant ventilation rate for the anticipated occupancy load. The ventilation rates for specified occupied spaces listed in Table 2 were selected to reflect the consensus that the provision of acceptable outdoor air at these rates would achieve an acceptable level of indoor air quality by reasonably controlling $CO_2$, particulates, odors, and other contaminants common to those spaces. (Appendix D shows the outdoor air needed to control occupant-generated $CO_2$ under various conditions.)

Human occupants produce carbon dioxide, water vapor, particulates, biological aerosols, and other contaminants. Carbon dioxide concentration has been widely used as an indicator of indoor air quality. Comfort (odor) criteria are likely to be satisfied if the ventilation rate is

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TABLE 2.3[a]
OUTDOOR REQUIREMENTS FOR VENTILATION OF RESIDENTIAL FACILITIES
(Private Dwellings, Single, Multiple)

| Applications | Outdoor Requirements | Comments |
|---|---|---|
| Living areas | 0.35 air changes per hour but not less than 15 cfm (7.5 L/s) per person | For calculating the air changes per hour, the volume of the living spaces shall include all areas within the conditioned space. The ventilation is normally satisfied by infiltration and natural ventilation. Dwellings with tight enclosures may require supplemental ventilation supply for fuel-burning appliances, including fireplaces and mechanically exhausted appliances. Occupant loading shall be based on the number of bedrooms as follows: first bedroom, two persons; each additional bedroom, one person. Where higher occupant loadings are known, they shall be used. |
| Kitchens[b] | 100 cfm (50 L/s) intermittent or 25 cfm (12 L/s) continuous or openable windows | Installed mechanical exhaust capacity[c]. Climatic conditions may affect choice of the ventilation system. |
| Baths, Toilets[b] | 50 cfm (25 L/s) intermittent or 20 cfm (10 L/s) continuous or openable windows | Installed mechanical exhaust capacity[c]. |
| Garages: Separate for each dwelling unit | 100 cfm (50 L/s) per car | Normally satisfied by infiltration or natural ventilation |
| Common for several units | 1.5 cfm/ft$^2$ (7.5 L/s·m$^2$) | See "Enclosed parking garages," Table 2.1 |

[a] In using this table, the outdoor air is assumed to be acceptable.
[b] Climatic conditions may affect choice of ventilation option chosen.
[c] The air exhausted from kitchens, bath, and toilet rooms may utilize air supplied through adjacent living areas to compensate for the air exhausted. The air supplied shall meet the requirements of exhaust systems as described in 5.8 and be of sufficient quantities to meet the requirements of this table.

set so that 1000 ppm $CO_2$ is not exceeded. In the event $CO_2$ is controlled by any method other than dilution, the effects of possible elevation of other contaminants must be considered (see Refs 12-18).

**6.1.3.1 Multiple Spaces.** Where more than one space is served by a common supply system, the ratio of outdoor to supply air required to satisfy the ventilation and thermal control requirements may differ from space to space. The system outdoor air quantity shall then be determined using Equation 6-1 (see Refs 24 and 25).

$$Y = X/[1 + X - Z] \qquad (6\text{-}1)$$

where

$Y = V_{ot}/V_{st}$ = corrected fraction of outdoor air in system supply
$X = V_{on}/V_{st}$ = uncorrected fraction of outdoor air in system supply
$Z = V_{oc}/V_{sc}$ = fraction of outdoor air in critical space. The critical space is that space with the greatest required fraction of outdoor air in the supply to this space.
$V_{ot}$ = corrected total outdoor air flow rate
$V_{st}$ = total supply flow rate, i.e., the sum of all supply for all branches of the system
$V_{on}$ = sum of outdoor air flow rates for all branches on system
$V_{oc}$ = outdoor air flow rate required in critical spaces
$V_{sc}$ = supply flow rate in critical space

Equation 6-1 is plotted in Fig. 3. The procedure is as follows:

1. Calculate the uncorrected outdoor air fraction by dividing the sum of all the branch outdoor air requirements by the sum of all the branch supply flow rates.

2. Calculate the critical space outdoor air fraction by dividing the critical space outdoor air requirement by the critical space supply flow rate.

3. Evaluate Equation 6-1 or use Fig. 3 to find the corrected fraction of outdoor air to be provided in the system supply.

Rooms provided with exhaust air systems, such as kitchens, baths, toilet rooms, and smoking lounges, may utilize air supplied through adjacent habitable or occupiable spaces to compensate for the air exhausted. The air supplied shall be of sufficient quantity to meet the requirements of Table 2. In some cases, the number of persons cannot be estimated accurately or varies considerably. In other cases, a space may require ventilation to remove contamination generated within the space but unrelated to human occupancy (e.g., outgassing from building materials or furnishings). For these cases, Table 2 lists quantities in cfm/ft$^2$ (L/s·m$^2$) or an equivalent term. If human carcinogens or other harmful contaminants are suspected to be present in the occupied space, other relevant standards or guidelines (e.g., OSHA, EPA) must supersede the ventilation rate procedure.

When spaces are unoccupied, ventilation is not generally required unless it is needed to prevent accumulation of contaminants injurious to people, contents, or structure. Design documentation shall specify all significant assumptions about occupants and contaminants.

**6.1.3.2 Recirculation Criteria.** The requirements for ventilation air quantities given in Table 2 are for 100% outdoor air when the outdoor air quality meets the specifications for acceptable outdoor air quality given in 6.1.1. While these quantities are for 100% outdoor air, they also set the amount of air required to dilute contaminants to acceptable levels. Therefore, it is necessary that at least this amount of

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air be delivered to the conditioned space at all times the building is in use except as modified in 6.1.3.4.

Properly cleaned air may be recirculated. Under the ventilation rate procedure, for other than intermittent variable occupancy as defined in 6.1.3.4, outdoor air flow rates may not be reduced below the requirements in Table 2. If cleaned, recirculated air is used to reduce the outdoor air flow rate below the values shown in Table 2, the Air Quality Procedure, 6.2, must be used. The air-cleaning system for the recirculated air may be located in the recirculated air or in the mixed outdoor and recirculated airstream (see Fig. 1).

The recirculation rate for the system is determined by the air-cleaning system efficiency. The recirculation rate must be increased to achieve full benefit of the air-cleaning system. The air-cleaning used to clean recirculated air should be designed to reduce particulate and, where necessary and feasible, gaseous contaminants. The system shall be capable of providing indoor air quality equivalent to that obtained using outdoor air at a rate specified in Table 2. Appendix E may be referenced for assistance in calculating the air flow requirements for commonly used air distribution systems.

**6.1.3.3 Ventilation Effectiveness, $E_v$:** Outdoor air for controlling contaminant concentration can be used for dilution or for sweeping the contaminants from their source. The values in Table 2 define the outdoor air needed in the occupied zone for well-mixed conditions (ventilation effectiveness approaches 100%). The ventilation effectiveness is defined by the fraction of the outdoor air delivered to the space that reaches the occupied zone.

Ventilation effectiveness may be increased by creating a plug flow situation. If the flow pattern is such that the ventilation air flows past the contaminant source and sweeps the contaminant toward an exhaust, the contaminant concentration in the exhaust can be greater than that for the well-mixed condition. Ventilation effectiveness can then be greater than that which would be realized with perfect mixing. Local exhaust systems operate in this way. With perfect mixing between the ventilation air and the air in a space, ventilation effectiveness is 100%. With perfect mixing, $E_v = 1.0$. It is, however, not uncommon to find some of the ventilation air bypassing the occupants (moving from supply to exhaust without fully mixing in the occupied zone) and achieving $E_v$ values as low as 0.5 (see Ref 26). Such flow conditions should be avoided. The ability of the ventilation air to mix in the occupied zone can be improved through recirculation or active mixing of the air in the space. Additional information about ventilation effectiveness can be found in Appendix F.

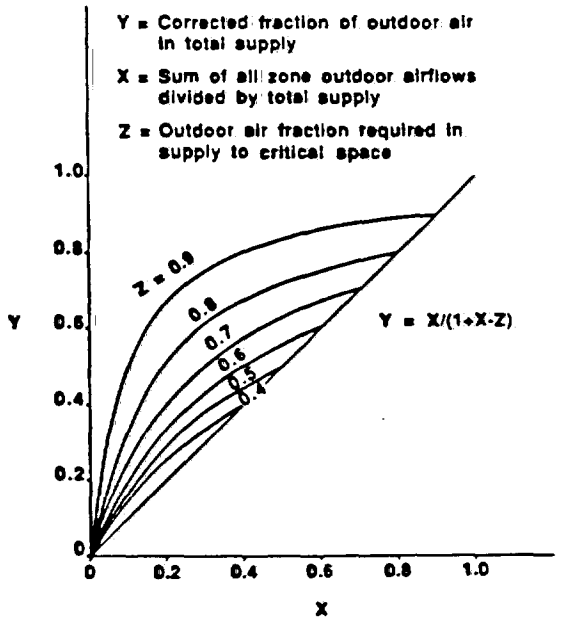


*Fig. 3 Ventilation Reduction in Multiple Spaces Supplied from a Common Source*

**6.1.3.4 Intermittent or variable occupancy.** Ventilating systems for spaces with intermittent or variable occupancy may have their outdoor air quantity adjusted by use of dampers or by stopping and starting the fan system to provide sufficient dilution to maintain contaminant concentrations within acceptable levels at all times. Such system adjustment may lag or should lead occupancy depending on the source of contaminants and the variation in occupancy. When contaminants are associated only with occupants or their activities, do not present a short-term health hazard, and are dissipated during unoccupied periods to provide air equivalent to acceptable outdoor air, the supply of outdoor air may lag occupancy. When contaminants are generated in the space or the conditioning system independent of occupants or their activities, supply of outdoor air should lead occupancy so that acceptable conditions will exist at the start of occupancy. Figures 4 and 5 show lag or lead times needed to achieve acceptable conditions for transient occupancy (see Appendix G for rationale). Where peak occupancies of less than three hours duration occur, the outdoor air flow rate may be determined on the basis of average occupancy for buildings for the duration of operation of the system, provided the average occupancy used is not less than one-half the maximum. Caution should be exercised for spaces that are allowed to lag and may be affected, due to pressure differences, by contaminants entering from adjacent spaces, such as parking garages, restaurants, etc.

**6.2 Indoor Air Quality Procedure:** This procedure provides an alternative performance method to the Ventilation Rate Procedure for achieving acceptable air quality. The Ventilation Rate Procedure described in 6.1 is deemed to provide acceptable indoor air quality, ipso facto. Nevertheless, that procedure, through prescription of required ventilation rates, provides only an indirect solution to the control of indoor contaminants. The Indoor Air Quality Procedure provides a direct solution by restricting the concentration of all known contaminants of concern to some specified acceptable levels. It incorporates both quantitative and subjective evaluation.

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**6.2.1 Quantitative Evaluation.** Table 1 furnishes information on acceptable contaminant levels in outdoor air. This table also applies indoors for the same exposure times. For additional information on contaminants in the outdoor air, see 6.1.1. Table 3 contains limits for four other indoor contaminants. Three of these are limits set by other bodies as indicated in the table. The limit for $CO_2$ was selected based on the rationale outlined in Appendix D. Other potential contaminants for which definite limits have not been set are discussed in Appendix C. Tables C-1 and C-3 do not include all known contaminants that may be of concern, and these concentration limits may not, ipso facto, ensure acceptable indoor air quality with respect to other contaminants.

Human occupants produce carbon dioxide, water vapor, particulates, biological aerosols, and other contaminants. Carbon dioxide concentration has been widely used as an indicator of indoor air quality. A limit of 1000 ppm $CO_2$ is recommended to satisfy comfort (odor) criteria. In the event $CO_2$ is controlled by any method other than dilution, the effects of the possible elevation of other contaminants must be considered.

In recent years a number of indoor contaminants have received increased attention and emphasis. Some of these contaminants, such as formaldehyde or other vapor phase organic compounds, are generated by the building, its contents, and its site. Another important group of contaminants is produced by unvented indoor combustion. The presence and use of consumer and hobby products, as well as cleaning and maintenance products, introduce a range of largely episodic releases of contaminants to the indoor environment (see Ref 30).

There are also complex mixtures, such as environmental tobacco smoke (see Ref 31), infectious and allergenic biologic aerosols, emanations from human bodies, and

PROCEDURE

a. Compute the air capacity per person in the space in $ft^3$ ($m^3$).
b. Find the required ventilation rate, in cfm (L/s) per person.
c. Enter Figure 4 with these values and read the maximum permissible ventilation lag time after occupancy from the intersection of a and b.

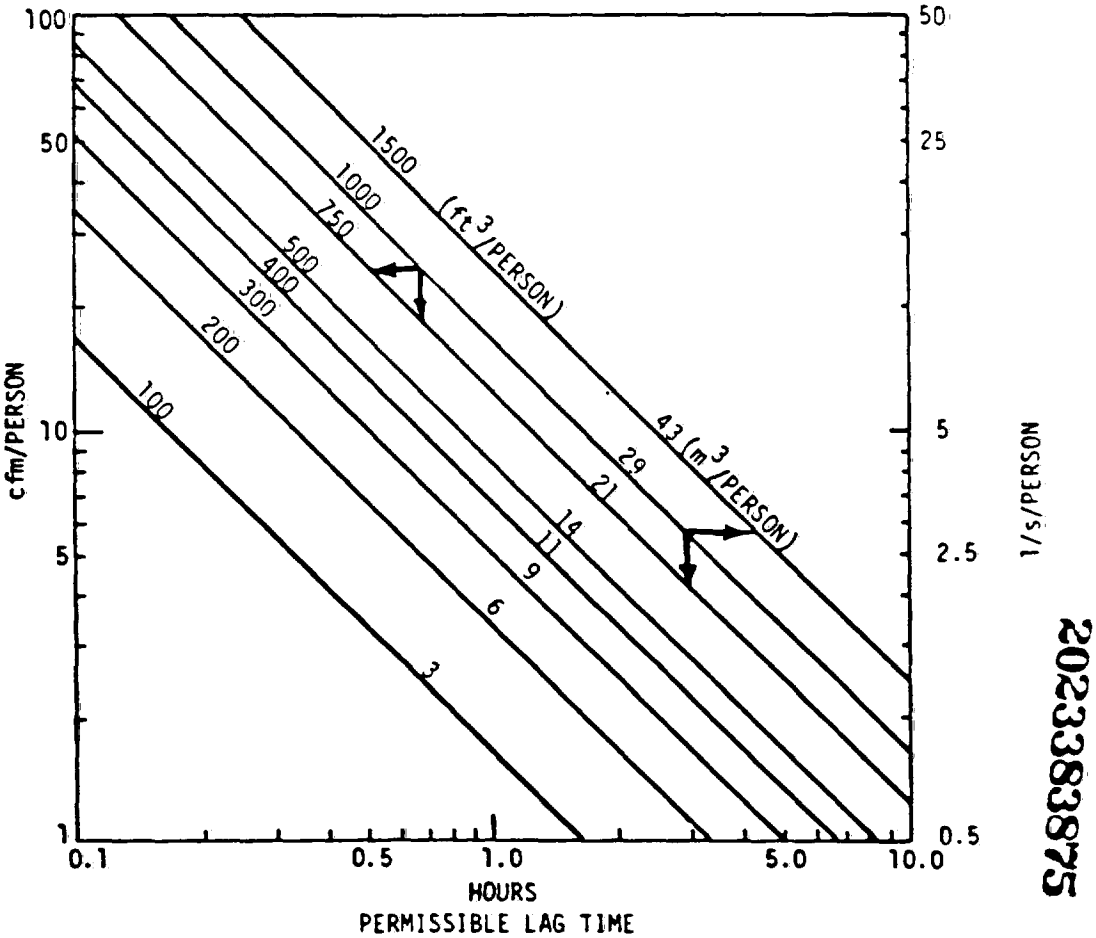


*Fig. 4 Maximum Permissible Ventilation Lag Time*

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TABLE 3
GUIDELINES FOR SELECTED AIR CONTAMINANTS OF INDOOR ORIGIN

| Contaminant | Concentration | ppm | Exposure Time | Comments |
|---|---|---|---|---|
| Carbon Dioxide | 1.8 g/m³ | 1000* | Continuous | See Appendix D |
| Chlordane | 5 ug/m³ | 0.0003 | Continuous | Reference 27 |
| Ozone | 100 ug/m³ | 0.05 | Continuous | Reference 28 |
| Radon gas | 0.027 WL | | Annual Average | Reference 29, Paragraph 12.6 (background 0.002- 0.994WL) |

*This level is not considered a health risk but is a surrogate for human comfort (odor). See Section 6.1.3 and Appendix D.

emanations from food preparation. Precise quantitative treatment of these contaminants can be difficult. To some degree, adequacy of control must rest upon subjective evaluation.

In the case of some odorless biologic aerosols, subjective evaluation is irrelevant. Application of generally acceptable technology, and vigilance regarding adverse influences of reduced ventilation, must therefore suffice. Appendix C contains information on standards and guidelines for selected air contaminants. Uniform governmental policies regarding limits on exposure to environmental carcinogens have not yet emerged.

PROCEDURE

a. Compute the air capacity per person in the space in ft³ (m³).
b. Find the required ventilation rate, in cfm (L/s) per person.
c. Enter Figure 5 with these values and read the maximum permissible ventilation lag time after occupancy from the intersection of a and b.

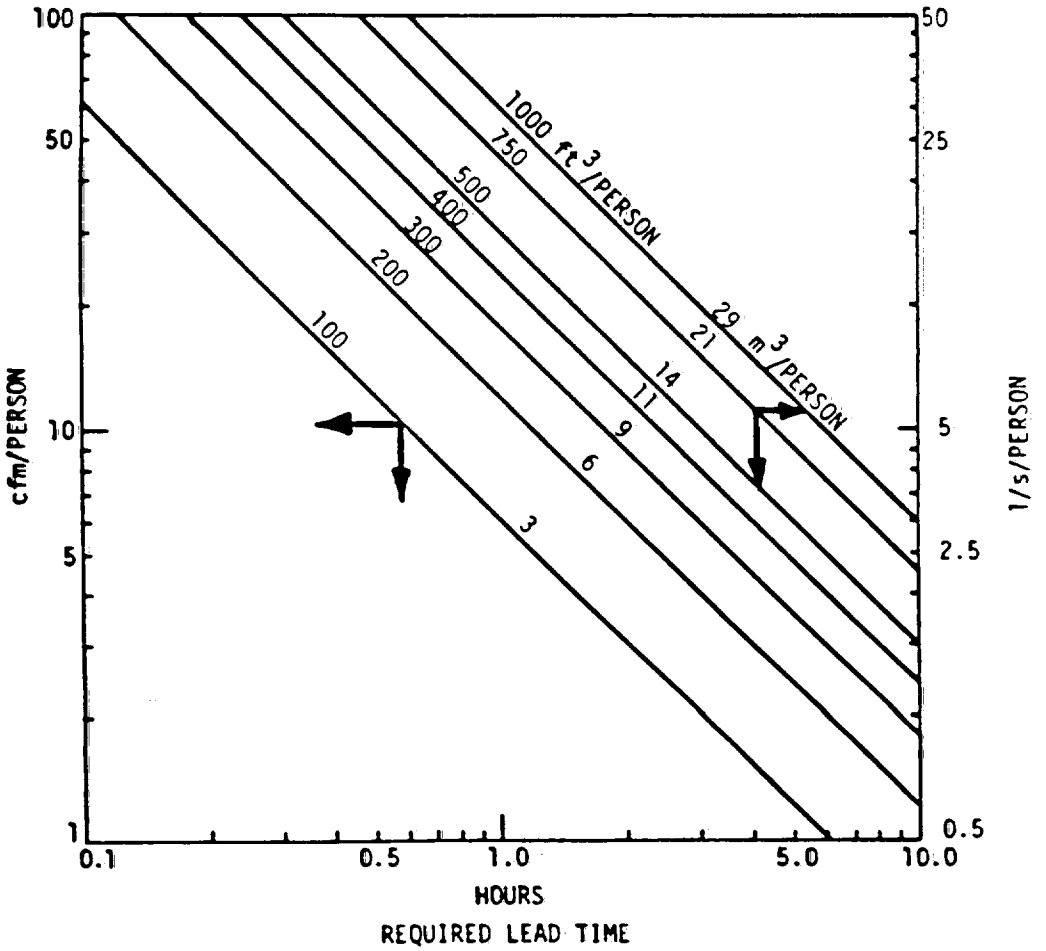


*Fig. 5 Minimum Ventilation Time Required Before Occupancy of Space*

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**6.2.2 Subjective Evaluation.** Various indoor air contaminants may give rise to odor that is of unacceptable intensity or character or that irritates the eyes, nose, or throat. In the absence of objective means to assess the acceptability of such contaminants, the judgment of acceptability must necessarily derive from subjective evaluations of impartial observers. One method that may be used for measuring subjective response is described in Appendix C. Caution should be used in any subjective evaluation procedure to avoid unacceptable concentrations of other contaminants.

**6.2.3 Air Cleaning.** Recirculation criteria are defined in 6.1.3.2 for use with the Ventilation Rate Procedure. Recirculation with air-cleaning systems is also an effective means for controlling contaminants when using the Indoor Air Quality Procedure. The allowable contaminant concentration in the occupied zone can be used with the various system models in Appendix E to compute the required outdoor air flow rate. The air-cleaning system efficiency for the troublesome contaminants present, both gaseous and particulate, may be adequate to satisfy the Indoor Air Quality criteria of 6.2.1 and 6.2.2. However, contaminants that are not appreciably reduced by the air-cleaning system may be the controlling factor in design and prohibit the reduction of air below that set by the Ventilation Rate Procedure.

**6.3 Design Documentation Procedures.** Design criteria and assumptions shall be documented and should be made available for operation of the system within a reasonable time after installation. See Sections 4 and 6 as well as 5.2 and 6.1.3 regarding assumptions that should be detailed in the documentation.

## 7. REFERENCES


[1] *ANSI/ASHRAE Standard 55-1981, Environmental Conditions For Human Occupancy.* American Society of Heating, Refrigerating, and Air-Conditioning Engineers, Inc., Atlanta, GA 30329. 1981.

[2] *UL 181, Factory Made Air Ducts and Air Duct Connectors,* March 19, 1984. Underwriters' Laboratories, 333 Pfingsten Rd., Northbrook, IL 60611. 1984.

[3] *NFPA 90A 1978, Standard for the Installation of Air Conditioning and Ventilating Systems.* National Fire Protection Association, Battery March Park, Quincy, MA 02269. 1978.

[4] *NFPA 90B 1980, Standard for the Installation of Warm Air Heating and Air Conditioning Systems.* National Fire Protection Association, Battery March Park, Quincy, MA 02269. 1980.

[5] SMACNA Duct Construction Standards: *High-pressure Duct Construction Standards,* third ed., 1975; *Low-pressure Duct Construction Standards,* fifth ed., 1976. Sheet Metal and Air-Conditioning Contractors National Association, Inc., 8224 Old Courthouse Road, Tyson Corners, Vienna, VA 22180. 1975, 1976.

[6] SMACNA. 1979. *Fiberous Glass Duct Construction,* Fifth ed. Sheet Metal and Air-Conditioning Contractors National Association Inc., 8224 Old Courthouse Road, Tyson Corners, Vienna, VA 22180. 1979.

[7] ACGIH. 1986. *Industrial Ventilation—A Manual of Recommended Practice—1986 ed.* American Conference of Governmental Industrial Hygienists, Committee on Industrial Ventilation, P.O. Box 16153, Lansing, MI 48901. 1986.

[8] *ASHRAE Standard 52-76, Method of Testing Air Cleaning Devices Used in General Ventilation for Removing Particulate Matter.* American Society of Heating, Refrigerating, and Air-Conditioning Engineers, Inc., Atlanta, GA 30329. 1976.

[9] U.S., Department of Defense. *MIL Standard 282 Filter Units, Protective Clothing, Gas-Masks.* Available from Global Engineering, Documents, P.O. Box 2504, 2625 Hickory St., Santa Ana, CA 92707.

[10] *ASHRAE Handbook—1983 Equipment Volume,* Chapter 11, Table 1. American Society of Heating, Refrigerating, and Air-Conditioning Engineers, Inc., Atlanta, GA 30329. 1983.

[11] Sterling, E.M., A. Arundel, T.D. Sterling. 1985. "Criteria for human exposure to humidity in occupied buildings." *ASHRAE Transactions,* Vol. 91, Part 1B, pp. 611-622.

[12] Janssen, J.E., and A. Wolff. 1986. "Subjective response to ventilation." In *Managing Indoor Air for Health and Energy Conversation, Proceedings of the ASHRAE Conference IAQ '86.* Atlanta: American Society of Heating, Refrigerating, and Air-Conditioning Engineers, Inc.

[13] Raijhans, G.S. 1983. "Indoor air quality and $CO_2$ levels. *Occupational Health in Ontario* 4:160-167.

[14] Berg-Munch, B., Clausen, B.G., and P.O. Fanger. 1984. "Ventilation requirements for the control of body odor in space occupied by women." In *Environment International,* Vol. 12 (1986), pp. 195-199.

[15] Leaderer, B.P. and W. Cain. 1983. "Air quality in buildings during smoking and non-smoking occupancy." *ASHRAE Transactions,* Vol. 89, Part 2B, pp. 601-613.

[16] Thayer, W.W.. 1982. "Tobacco smoke dilution recommendations for comfortable ventilation." *ASHRAE Transactions,* Vol. 88, Part 2, pp. 291-306.

[17] Bell, S.J. and B. Khati. 1883. "Indoor air quality in office buildings. *Occupational Health in Ontario,* 4:103-118.

[18] Hicks, J.. 1984. "Tight building syndrome:: When work makes you sick. *Occupational Health and Safety,* Jan. pp. 51-56.

[19] *National Primary and Secondary Ambient Air Quality Standards, Code of Federal Regulations, Title 40 Part 50 (40 CFR50).* U.S. Environmental Protection Agency.

[20] Morey, P.R., W.G. Jones, J.L. Clere, and W.G. Sorenson. 1986. "Studies on sources of airborne microorganisms and on indoor air quality in a large office building. In *Managing Inddor Air for Health and Energy Conservation, Proceedings of the ASHRAE Conference IAQ '86,* pp. 500-509. Atlanta: American Society of Heating, Refrigerating, and Air-Conditioning Engineers, Inc.

[21] *NIOSH Manual of Analytical Methods,* 2nd Ed., April 1977. Publ. No. 77-157, 4 vols. Cincinnati: National Institute for Occupational Safety and Health.

[22] *NIOSH Manual Sampling Data Sheets* with Suppl., Pubs. Nos. 77-159 and 78-189, March, 1977 and August, 1978. Note: The Clearinghouse for Occupational Safety and Health of NIOSH, 4676 Columbia Parkway, Cincinnati, OH 45226, is willing to fill occasional requests for separate sheets of the information on individual air contaminants from these publications on request. National Institute for Occupational Safety and Health, Cincinnati, OH. 1978.

[23] *ACGIH Threshold Limit Values and Biological Exposure Indices for 1985-86.* 1985. Cincinnati: American Conference of Governmental Industrial Hygienists, 6500 Glenway, Bldg. D-7, 45211-4438.

[24] Standards Association of Australia. 1980. *Australian Standard AS1668 Part 2, 1980—Ventilation Requirements,* Clause 3.5.2, Appendix A&B. Standards Association of Australia, Standards House, 80 Arthur St., North Sydney, NSW, 2060. 1980.

[25] Kowalczewski, J.J. 1973. "Quality of air in air conditioning." *AIRAH,* Feb. Australian Institute of Refrigeration, Air Conditioning and Heating.

[26] Janssen, J.E., T. Hill, J.E. Woods, and E.A.B. Maldonado. 1982. "Ventilation for control of indoor air quality: A case study?" *Environment International,* El 8 487-496.


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[27] U.S. National Academy of Sciences Committee on Toxicology. *An Assessment of Health Risk of Seven Pesticides Used in Termite Control, (Chlordane in Military Housing).* August 1979.

[28] *Maximum Acceptable Level of Ozone. Code of Federal Regulations.* Title 21, Part 801.415. Food and Drug Administration, U.S. Department of Health, Education, and Welfare.

[29] *Exposure from the Uranium Series with Emphasis on Radon and Its Daughters.* 1984. NCRP Report #77. The National Council on Radiation Protection and Measurement, Bethesda, MD.

[30] *NAP 1981 Indoor Pollutants.* 1981. National Academy Press, Washington, DC.

[31] *The Consequences of Involuntary Smoking.* 1986. U.S. Surgeon General, U.S. Dept. of Health and Human Services.

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* Listed standards were referenced at the time of public review in September 1986. The users may wish to refer to more current issues of these referenced standards.

**This Appendix is not part of this Standard but is included for information purposes only.**

## APPENDIX A

### CONVERSION FACTORS (A-1)

**Parts per million and mass per unit volume**

Measurement of airborne concentration of substances is generally converted to 77 °F (25 °C) and 29.92 in. Hg (760 mm Hg) pressure. Vapors or gases are often expressed as parts per million (ppm) by volume and are also frequently expressed in units of mass per unit volume, commonly in the following units:

milligram per cubic meter ($mg/m^3$); microgram per cubic meter ($\mu g/m^3$);
milligram per cubic foot ($mg/ft^3$); grains per cubic foot ($gr/ft^3$).

The ppm values may be converted to mass per unit volume values as follows:

ppm × molecular weight/24,450 = mg/L
ppm × molecular weight/0.02445 = $\mu g/m^3$
ppm × molecular weight/24.45 = $mg/m^3$
ppm × molecular weight × 28.3/24,450 = $mg/ft^3$
ppm × molecular weight × 28.3/64.8/24,450 = $gr/ft^3$

Airborne particle count concentrations measured in million particles per cubic foot (mppcf) or million particles per cubic meter (= particles per cubic centimeter, cc) can be converted approximately to mass per unit volume as follows when density and mass median diameter have not been determined:

mppcf × 6 (approximately) = $mg/m^3$
particles per cc × 210 (approximately) = $mg/m^3$

**Units for Measuring Radon Progeny Concentrations and Exposures**

Airborne concentrations of radon progeny, like radon itself, can be specified in picocuries per liter (pCi/L) or equivalent units. For radiation protection purposes, it has been useful to characterize radon progeny concentrations in terms of the total alpha energy emitted as a result of decay of the short-lived progeny (polonium 218 to polonium 214) to lead 210, a long-lived radionuclide. This "potential alpha energy concentration" (PAEC) is an indicator of potential dose to the lung, which, in turn, may be associated with increased lung cancer incidence on the basis of epidemiological studies (see Ref A-2) and other evidence.

The conventional unit for PAEC is the working level (WL), which has a value of $1.3 \times 10^5$ MeV/L, the potential alpha energyper unit volume that would be associated with air containing approximately 100 pCi/L of each of the short-lived progeny. For an arbitrary mixture with polonium 218 concentration (1a), lead 214 concentration (1b), and bismuth 214 concentration (1c), the PAEC is approximately equal to (0.10 1a + 0.51 1b + 0.37 1c) (WL/100, pCi/L). The associated exposure unit, working level month (WLM), is the exposure that an individual would experience remaining in 1 WL of progeny for 173 hours (an average working month).

If a volume were to have a constant source of radon and no mechanisms (other than radioactive decay) for removal of radon or its progeny from the enclosed air, the activity concentrations of each radionuclide (given in pCi/L) would eventually reach a state where all were numerically equal. Such a condition (referred to as "equilibrium") is never achieved in practice because of removal mechanisms such as ventilation and progeny "plateout." Ventilation both reduces the radon concentration and decreases the ratio of progeny to their parents below one. Plateout, the attachment of progeny to walls and other surfaces, also decreases this ratio.

The equilibrium condition of radon and its progeny is conventionally indicated by an "equilibrium factor" (F) that is the ratio of actual progeny PAEC to the PAEC were each offspring to have the same activity concentration as that of the radon actually present. Thus F = PAEC/(radon concentration/100), where the PAEC is given in WL and the radon concentration in pCi/L. In spaces with low progeny-removal rates, F is close to one. In houses, equilibrium factors have usually been found to lie in the range of 0.2 to 0.8, although factors above and below this range have sometimes been found. Taking 0.5 as a typical equilibrium factor, the annual exposure associated with a constant radon concentration of 1 pCi/L may be calculated as follows:

exposure rate for 1 pCi/L =

$$0.5\ (1\ \text{pCi/L}) \left(\frac{1\ \text{WL}}{100\ \text{pCi/L}}\right) \left(\frac{1\ \text{WLM}}{1\ \text{WL} \times 173\ \text{h}}\right) \left(\frac{8760}{\text{year}}\right)$$

$$= 0.25\ \text{WLM/year}$$

Also, 1 pCi/L = 37 $Bq/m^3$

**References for Appendix A**


A-1. *Conversion Units and Factors Relating to Atmospheric Analysis, Recommended practice for ASTM-D-1914-68.* American Society of Testing and Materials, 1916 Race Street, Philadelphia, PA 19103. 1983.

A-2. *Exposure from the Uranium Series with Emphasis on Radon and Its Daughters,* NCRPM Report #77. The National Council on Radiation Protection and Measurement, Washington, DC.


**This Appendix is not part of this Standard but is included for information purposes only.**

## APPENDIX B

### POSITIVE COMBUSTION AIR SUPPLY

Fuel-fired appliances equipped with an open draft hood for control of combustion chamber draft must exhibit a positive flow of air into the draft hood whenever combustion is present. Measurements are made when building infiltration is low, i.e., the inside-outside temperature difference should be no more than 30 °F (18 °C) and wind velocity is no more than 5 mph (2.2 m/s). Commonly used exhaust fans, such as kitchen and bathroom exhaust fans, should be turned on, and fireplaces that have no dedicated combustion air supply should be operated simultaneously with all external doors and windows closed. Flow of room air into the draft hood under these conditions must indicate a 40% dilution of the products of combustion going up the stack (see Ref B-1). The dilution ratio for products of combustion can be determined by measuring the room air temperature entering the draft hood, the stack temperature downstream from the draft hood, and the flue temperature at the combustion chamber outlet just upstream of the draft hood before the draft hood dilution air cools the flue gas. Then

$$(T_f - T_s)/(T_s - T_r) = 0.40$$

$T_f$ = flue temperature
$T_s$ = stack temperature
$T_r$ = room temperature

If the stack temperature exceeds

$$T_s = (T_f + 0.4T_r)/1.4$$



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under the measurement conditions defined above, a positive supply of outside combustion air is needed for safe operation of the furnace. Reliable measurements of the stack temperature may be difficult because of nonuniformities across the stack due to incomplete mixing. (Methods based on pressure measurements can also be used.) Cold stack conditions may cause special problems (see Ref B-2).

Power burners have a blower to supply combustion air. There must be enough air supplied to this type of burner to ensure that the burner blower produces the pressure rise specified by the manufacturer. If a building is so tight that the blower cannot achieve its rated pressure rise, a positive supply of outdoor air must be provided. Care must be exercised with oil burners, however, if cold outdoor air is ducted directly to the burner. The low air temperature may degrade atomization and burner efficiency. The outdoor air provided should be tempered by heat loss from the stack and furnace jacket.

## REFERENCES


B-1. *ASHRAE Handbook—1983 Equipment Volume*, Chapter 27, p. 27.24. American Society of Heating, Refrigerating, and Air-Conditioning Engineers, Inc., Atlanta, GA 30329. 1983.

B-2. *Residential Combustion Venting Failure: A Systems Approach*. Research Div., Canada Mortgage and Housing Corp, 682 Montreal Rd., Ottawa, Canada, K1A OP7, July 30, 1987, 145 pp. 1987.


**This Appendix is not part of this Standard but is included for information purposes only.**

## APPENDIX C
## GUIDANCE FOR THE ESTABLISHMENT OF AIR QUALITY CRITERIA FOR THE INDOOR ENVIRONMENT

The ventilation rates recommended in Table 2 are based on existing practice in indoor environments that contain the specified occupant density and for activities that can normally be expected to take place in such environments. Whenever building materials, cleaning and maintenance materials, or specialized human activities introduce large quantities of specific contaminants into the building atmosphere there may be occupant complaints and special measures should be considered to alleviate them.

*Threshold Limit Values for Chemical Substances in the Work Environment Adopted by ACGIH* is obtainable from the Publications Office, American Conference of Governmental Industrial Hygienists, 6500 Glenway Avenue, Building D-7, Cincinnati, OH 45211-4438 (see Ref C-1; Ref C-2 is the West German counterpart). This publication provides 8-hour, 15-minute, and instantaneous case limits. It is a source of concentration limits for many chemical substances and physical agents for industrial hygiene use. In light of the constantly changing state of knowledge, the document is updated annually. It cautions the user, "The limits listed in this book are intended for use in the practice of industrial hygiene as guidelines or recommendations in the control of potential health hazards and for no other use."

Industrial health practice attempts to limit worker exposure to injurious substances at levels that do not interfere with the work process and do not injure the workers' health. The elimination of all effects, e.g., unpleasant smells or mild irritation, is not attempted. Regulations are based on the results of accumulated experience with worker health and of animal experiments, carefully evaluated by groups of competent experts. Exposure and effects are related to dose of the injurious substance. Dose includes both the concentration of the substance and the time during which it is present. Since concentration commonly varies with time, dose is conveniently expressed as a time, weighted average concentration (TWA), short-term exposure limit (STEL), or threshold limit value (TLV). Regulations of the U.S. Occupational Safety and Health Administration are TWAs in most cases. Industrial exposures are regulated on the basis of a 40-hour work week with 8- to 10-hour days. The remainder of the time exposure is anticipated to be substantially lower for the pollutant of concern. For contaminants where standards or guidelines have not been established, it has been customary to assume as a first guide that a concentration of 1/10 TLV would not produce complaints in a nonindustrial population in residential, office, school, or other similar environments. The 1/10 TLV may not provide an environment satisfactory to individuals who are extremely sensitive to an irritant. In any event, where standards or guidelines do not exist, expert help should be sought in evaluating what level of such a chemical or combination of chemicals would be acceptable.

Guidelines have been established for a number of chemicals and metals that may be found in the outdoor air, as shown in Refs C-3 through C-9. Most would normally be found only in areas near certain industrial facilities, but some may be found in residential areas. These references are offered as sources of information when the quality of the outdoor air is suspect.

Tables C-1 and C-2 present lists of North American standards and guidelines for acceptable concentrations of substances in the indoor and outdoor environment. Table C-3 presents a summary of Canadian exposure guidelines for residential indoor air quality. Table C-4 presents a list of substances evaluated by a working group on indoor air quality research of the World Health Organization. These tables are presented as further background information when using the Indoor Air Quality Procedure.

Many contaminants have odors or are irritants that may be detected by human occupants or visitors to a space. The air can be considered acceptably free of annoying contaminants if 80% of a panel of at least 20 untrained observers deems the air to be not objectionable under representative conditions of use and occupancy. An observer should enter the space in the manner of a normal visitor and should render a judgment of acceptability within 15 seconds. Each observer should make the evaluation independently of other observers and without influence from a panel leader. Users of this method are cautioned that the method is only a test for odors. Many harmful contaminants will not be detected by this test. Carbon monoxide and radon are two examples of odorless contaminants.

### References for Appendix C


C-1. *TLVs Threshold Limit Values and Biological Exposure Indices for 1986-87*. American Conference of Governmental Industrial Hygienists, 1986, 6500 Glenway, Building D-7, Cincinnati, OH 45211-4438. (Airborne concentrations of substances to which nearly all workers may be repeatedly exposed, day after day, without adverse effect; updated yearly.) 1986.

C-2. *Verein Deutscher Ingenieure, Handbuch Reinhaltung der Luft*. Maximale Imissions-Werte, VDI 2310, September 1974. (West German counterpart of TLVs at Ref C-1.

C-3. Newill, V.A. *Air Quality Standards*, Table III, pp. 462-487, in Vol. V of Stern, A.C. (ed.), *Air Pollution*, 3rd ed. Academy Press, New York, NY (national, by county, ambient air quality standards). 1977.

C-4. Government of Ontario, Regulation 296 under the Environmental Protection Act, Revised Regulations of Ontario, Toronto (current update of Ontario, Canada, ambient air quality criteria).

C-5. Martin, W., and A.C. Stern, *The World's Air Quality Standards*, Vol. II. *The Air Quality Management Standards of the United States*, Table 17, pp. 11-38, October 1974 (available from *NTIS PB-241-876*; National Technical Information Service, 5285 Port Royal Road, Springfield, VA 22161). 1974.

C-6. U.S. National Academy of Sciences, Committee on Toxicology, National Research Council, Guides for Short-Term Exposure of the Public to Air Pollutants. Microfiche or photocopies of these may be obtained from the National Technical Information Services, by order number. For example: *Ammonia PB-244-336, November 1972; Hydrochloric Acid PB-203-464, August 1971.*

C-7. U.S. Environmental Protection Agency, Code of Federal Regulations, Title 40, Part 61 (current national emission standards for hazardous air pollutants).

C-8. U.S. Environmental Protection Agency, National Air Toxics Information Clearinghouse Data Base, Report on State and Local Agency Air Toxics Activities, July 6, 1986 (tabulation of reporting states and communities published standards and guidelines for toxic air pollutants) 1986.

C-9. U.S. Environmental Protection Agency, Code of Federal Regulations, Title 40, Part 50 (current national ambient air quality standards).


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## TABLE C-1
## STANDARDS APPLICABLE IN THE UNITED STATES FOR COMMON INDOOR AIR POLLUTANTS[a]

| Pollutant | Indoor Standards | Outdoor Standards | Industrial Workplace Standards |
|---|---|---|---|
| Asbestos | Consumer Product Safety Commission has banned use of asbestos in artificial logs for fireplaces, in patching compounds, and in certain garments (16 CFR 1304, 1305, 1500.17(a)(7)); voluntary ban on use in hairdryers (C-10)<br><br>EPA regulates use in schools (40 CFR 763.80) and removal projects (40 CFR 763.120); bans installation of friable asbestos for facility insulation (40 CFR 61.150) (C-11) (C-7) | National Emissions Standard: no visible emissions; may also comply by cleaning emissions as specified before particulate asbestos material escapes to air (EPA, 40 CFR 61.140 et seq.) (C-7)<br><br>State air quality limits:<br><br>CT 0.0010 ug/m³ 8 hr.<br>MA 0.0001 fb/cm³ 24 hr.<br>NC 0.0100 ug/m³ 24 hr.<br>NY 5.0000 ug/m³ 1 yr.<br>VA 2.0000 ug/m³ 24 hr.<br>(all TLV based-fibers longer than 5 microns)[a]<br>(NATICH Data Base, 1986) (C-8) | 0.2 fiber/cm³ 8 hr. TWA (optically measured longer than 5 microns)(OSHA, 29 CFR 1910.1001(c) (C-12)<br><br>2.0 fiber/ml, 8 hr. TWA<br><br>(Mine Safety and Health Admin., 30 CFR (56.5001(b), 57.5001(b)) (C-13) |
| Carbon Monoxide (See Table 1) | | National Ambient Air Quality Primary Standard:<br><br>10 mg/m³ (9 ppm) 8 hr. avg.<br>40 mg/m³ (35 ppm) 1 hr. avg.<br><br>(EPA, 40 CFR 50.8) (C9)<br><br>State air quality limits:<br><br>CT 10000. ug/m³ 8 hr.<br>NV 1.3100 mg/m³ 8 hr.<br><br>(NATICH Data Base, 1986) (C-8) | 55 mg/m³ (50 ppm) 8 hr. TWA<br><br>(OSHA, 29 CFR 1910.1000, Table Z-1) (C-12)<br><br>Mine Safety and Health Admin. uses ACGIH TLV[a]<br><br>(30 CFR 57.5001(a)) (C-13) |
| Formaldehyde | Federal: 0.4 ppm target ambient level; HUD standard for manufactured homes, achieved through product emissions standards of .2 and .3 ppm (HUD, 24 CFR 3280.308, 1984) (C-14)<br><br>State: 0.4 ppm standard for indoor exposure (MN statute 144.495, 1985) (C-15) | No federal standard.<br>State air quality limits:<br><br>CT 12.00 ug/m³ 8 hr.<br>IL 0.0150 ug/m³ 1 yr.<br>IN 18.000 ug/m³ 8 hr.<br>MA 0.2000 ug/m³ 24 hr.<br>NC 300.00 ug/m³ 15 min.<br>NV 0.0710 mg/m³ 8 hr.<br>NY 2.0000 ug/m³ 1 yr.<br>VA 12.000 ug/m³ 24 hr.<br><br>(NATICH Data Base, 1986) (C-8) | 1 ppm 8 hr. TWA PEL[b]<br>2 ppm 15-min. STEL[c]<br><br>(OSHA, 29 CFR 1910.1000, Table Z-2; OSHA issued a final rule Dec. 4, 1987 (52 FR 46168) lowering a previous standard to the above levels, which was effective on Feb. 2, 1988)<br><br>Mine Safety and Health Admin. uses ACGIH TLVs<br><br>(30 CFR 57.5001(a)) (C-13) |
| Lead (See Table 1) | CPSC has banned in paint for consumer use or use on consumer products (16 CFR 1303) (C-10) | National Ambient Air Quality Primary and Secondary Standard:<br><br>1.5 ug/m³ max. arithmetic mean over calendar qtr. (EPA, 40 CFR 50.12) (C-9)<br><br>State air quality limits:<br><br>CT 1.500 ug/m³ 8 hr.<br>IL 0.500 ug/m³ 24 hr.<br>MA 0.680 ug/m³ 24 hr.<br>NV 0.004 mg/m³ 8 hr.<br>VA 2.500 ug/m³ 24 hr.<br><br>(NATICH Data Base for lead powder, 1986) (C-8) | 50 ug/m³ 8 hr. TWA<br><br>(OSHA, 29 CFR 1910.1025(c)) (C-12)<br><br>Mine Safety and Health Admin. uses ACGIH TLV<br><br>(30 CFR 57.5001(a)) (C13) |
| Nitrogen Dioxide (See Table 1) | | National Ambient Air Quality<br><br>Primary and Secondary Standards:<br><br>100 ug/m³<br>(0.053)ppm annual arithmetic mean<br><br>(EPA, 40 CFR 50.11) (C-9)<br><br>State air quality limits:<br><br>CT 120.0 ug/m³ 8 hr.<br>NV 0.143 mg/m³ 8 hr.<br><br>(NATICH Data Base, 1986) (C-8) | (5 ppm) ceiling 9 mg/m³<br><br>(OSHA, 29 CFR 1910.1000, Table Z-1) (C12)<br><br>Mine Safety and Health Admin. uses ACGIH TLV<br><br>(30 CFR 57.5001(a)) (C-12) |

[a]Most ACGIH TLVs are referenced in western nations' standards, including Canada, Western Europe, and Australia.

[b]PEL—Permissible exposure limit

[c]STEL—Short-term exposure limit

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## TABLE C-1
## STANDARDS APPLICABLE IN THE UNITED STATES FOR COMMON INDOOR AIR POLLUTANTS[a] (*Concluded*)

| Pollutant | Indoor Standards | Outdoor Standards | Industrial Workplace Standards |
|---|---|---|---|
| Ozone (See Table 1 and 3) | FDA prohibits devices (e.g., germicides, deodorizers) that result in more than 0.05 ppm in occupied enclosed spaces such as homes, offices, or hospitals, or that result in any releases in places occupied by the ill or infirm (21 CFR 801.415) (C-16) | National Ambient Air Quality Primary and Secondary Standards:<br>235 ug/m³ (0.12 ppm) max. hourly avg<br>(EPA, 40 CFR 50.9) (C9)<br>State air quality limits:<br>CT 235.0 ug/m³ 1 hr.<br>NV 0.005 mg/m³ 8 hr.<br>(NATICH Data Base, 1986) (C-8) | 0.2 mg/m³ (0.1 ppm) 8 hr. TWA<br>(OSHA, 29 CFR 1910.1000, Table Z-1) (C12)<br>Mine and Safety and Health Admin. uses ACGIH TLV<br>(30 CFR and 57.5001(a)) (C-13) |
| Particulates (See Table 1) | | National Ambient Air Quality Primary Standard:<br>75 ug/m³ annual geom. mean 260 ug/m³ maximum 24 hr.<br>Secondary Standard:<br>60 ug/m³ annual geom. mean 150 ug/m³ maximum 24 hr.<br>(EPA, 40 CFR 50.6, 50.7) (C-9) | |
| Radon (See Table 3) | | National Emission Standard for Radon-222 emissions from underground uranium mines—requires bulkhead construction (EPA, 40 CFR 61.22) (C-7)<br>National Emissions Standard for Radionuclide Emissions (excluding Radon-220, 222) from DOE facilities, other federal facilities, and NRC licensed facilities:<br>25 mrem/y whole body<br>75 mrem/7 critical organ<br>(EPA, 40 CFR 61.92, 61.102) (C-7) | 1.0 WL radon progeny maximum 4 WLM radon progeny calendar year<br>(Mine Safety and Health Admin., 30 CFR 57.5038, 57.5039) (C-13) |
| Sulfur Dioxide (See Table 1) | | National Ambient Air Quality Primary Standard:<br>80 ug/m³ (0.03 ppm) annual arithmetic mean<br>365 ug/m³ (0.14 ppm) 24 hr.<br>Secondary Standard:<br>1300 ug/m³ (0.5 ppm) 3 hr.<br>(EPA, 40 CFR 50.4, 50.5) (C-9)<br>State air quality limits:<br>CT 860.0 ug/m³ 8 hr.<br>NV 0.119 mg/m³ 8 hr.<br>TN 1.200 ug/m³ 1 yr.<br>(NATICH Data Base, 1986) (C-8) | 13 mg/m³ (5ppm) 8 hr. TWA<br>(OSHA, 29 CFR 1910.1000, Table Z-1) (C12)<br>Mine Safety and Health Admin. uses ACGIH TLV (30 CFR 57.5001(a)) (C-13) |

[a]Most ACGIH TLVs are referenced in western nations' standards, including Canada, Western Europe, and Australia.

## TABLE C-2
## GUIDELINES USED IN THE UNITED STATES FOR COMMON INDOOR AIR POLLUTANTS

| Pollutant | Indoor Guidelines | Outdoor Guidelines | Industrial Workplace Guidelines |
|---|---|---|---|
| Asbestos | | | 0.2-2.0 fibers/cm³ 8 hr. TLV-TWA (depending on type of fiber) (fibers longer than 5 microns) (ACGIH, 1986-87) (C-1) |

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TABLE C-2
GUIDELINES USED IN THE UNITED STATES FOR COMMON INDOOR AIR POLLUTANTS (*Concluded*)

| Pollutant | Indoor Guidelines | Outdoor Guidelines | Industrial Workplace Guidelines |
|---|---|---|---|
| Carbon Monoxide | | | 55 mg/m³ (50 ppm) 8 hr. TLV-TWA<br>440 mg/m³ (400 ppm) 15 min. STEL<br>(ACGIH, 1986-87) (C-1) |
| Chlordane | NAS recommendation for military housing: (C-17)<br>5 μg/m³ maximum | | |
| Formaldehyde | | | 1.5 mg/m³ (1 ppm) 8 hr. TLV-TWA<br>3 mg/m³ (2 ppm) 15 min. STEL<br>(ACGIH, 1986-87) (C-1)<br>1.2 mg/m³ (1 ppm) 8 hr. TWA<br>2.5 mg/m³ (2 ppm) 15 min. STEL<br>(American Industrial Hygiene Assn., 1986) (C20)<br>NAS recommendations for manned spacecraft: (C-18)<br>1.0 mg/m³ (1.0 ppm) 60 min.<br>0.1 mg/m³ (0.1 ppm) 90 days<br>0.1 mg/m³ (0.1 ppm) 6 mo.<br>Navy Submarine Atmospheric Control Manual, levels set by Naval Research Laboratory: (C-19)<br>3.0 ppm 1 hour<br>1.0 ppm 24 hour<br>0.5 ppm 90 days |
| Lead Dust and Fumes | | | 0.15 mg/m³ 8 hr. TLV-TWA<br>(ACGIH, 1986-87) (C-1) |
| Nitrogen Dioxide | | | 6 mg/m³ (3 ppm) 8 hr. TLV-TWA<br>10 mg/m³ (5 ppm) 15 min. STEL<br>(ACGIH, 1986-87) (C-1)<br>NAS recommendation for manned spacecraft: (C-18)<br>4 mg/m³ (2.0 ppm) 60 min.<br>1.0 mg/m³ (0.5 ppm) 90 days<br>1.0 mg/m³ (0.5 ppm) 6 mo |
| Ozone | | | 0.2 mg/m³ (0.1 ppm) 8 hr. TLV-TWA<br>0.6 mg/m³ (0.3 ppm) 15 min. STEL<br>(ACGIH, 1986-87) (C-1) |
| Radon<br>(See Table 3) | EPA 1986 recommendation for homes:<br>4 p Ci/l or less-can be reached in most homes<br>At 4-20 pCi/l-take action to reduce within a few years<br>At 20-200 pCi/l-reduce within several months<br>At 200 pCi/l or above,reduce within several weeks or relocate until levels are reduced<br>(EPA, "A Citizen's Guide to Radon," August 1986) (C-21)<br>(EPA, "Radon Reduction Methods, A Homeowner's Guide," August 1986) (C22) | | |
| Sulfur Dioxide | | | 5 mg/m³ (2 ppm) 8 hr. TLVTWA<br>10 mg/m³ (5 ppm) 15 min. STEL<br>(ACGIH, 1986-87) (C-1)<br>NAS recommendation for manned spacecraft (C-18)<br>13 mg/m³ (5.0 ppm) 60 min.<br>3 mg/m³ (1.0 ppm) 90 days<br>3 mg/m³ (1.0 ppm) 6 mo. |

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## TABLE C-3*
## SUMMARY OF CANADIAN EXPOSURE GUIDELINES FOR RESIDENTIAL INDOOR AIR QUALITY

| Contaminant | Acceptable Exposure Ranges: ASTER[g] | ALTER[h] | Page |
|---|---|---|---|
| Aldehydes (total) | $\Sigma c_i/C_i<1$[a] | — | 16 |
| Carbon dioxide | — | × 6 300 mg/m³ (<3 500 ppm) | 17 |
| Carbon monoxide | <11 ppm—8 h[b]<br><25 ppm—1 h[b] | — | 18 |
| Formaldehyde | [c] | [d]Action Level 120 µg/m³ (0.10 ppm)<br>Target Level 60 µg/m³ (0.05 ppm) | 26 |
| Nitrogen dioxide | <480 µg/m³ (<0.25 ppm)—1 h | <100 µg/m³ (<0.05 ppm) | 19 |
| Ozone | <240 µg/m³ (<0.12 ppm)—1 h | — | 21 |
| Particulate matter[e] | <100 µg/m³—1 h | <40 µg/m³ | 21 |
| Sulphur dioxide | <1 000 µg/m³(<0.38 ppm)—5 m | <50 µg/m³ (<0.019 ppm) | 23 |
| Water vapor | 30-80% R.H.—summer<br>30-55% R.H.—winter[f] | — | 24 |
| Others See below[i] | Minimize exposure | Minimize exposure | 28-36 |

[a] $C_i$ = 120 µg/m³ (formaldehyde); 50 µg/m³ (acrolein); 9000 µg/m³ (acetaldehyde), and $C_i$ are respective concentrations measured over a five-minute period.
[b] Units given only in parts per million so that guidelines are independent of ambient pressure.
[c] See Aldehydes (total).
[d] Although the epidemiological studies conducted to date provide little convincing evidence that formaldehyde is carcinogenic in human populations, because of this potential, indoor levels should be reduced as much as possible.
[e] <2.5 µm mass median aerodynamic diameter—MMMD.
[f] Unless constrained by window condensation.
[g] ASTER—Acceptable short-term exposure range.
[h] ALTER—Acceptable long-term exposure range.
[i] Other contaminants of concern: biological agents; chlorinated hydrocarbons; pest control products; product aerosols; fibrous materials, lead; polycyclic aromatic hydrocarbons (PAHs); and tobacco smoke.

*From Ref C-23, p.41.

## TABLE C-4
## WHO WORKING GROUP CONSENSUS OF CONCERN ABOUT INDOOR AIR POLLUTANTS AT 1984 LEVELS OF KNOWLEDGE*

| Pollutant[a] | Concentrations[b] reported | Concentrations[b] of limited or no concern | Concentraton[b] of concern | Remarks |
|---|---|---|---|---|
| Tobacco smoke (passive smoking)[i] | | | | |
| Respirable particulates | 0.05-0.7 | <0.1 | >0.15 | Japanese standard 0.15 mg/m |
| CO | 1-1.5 | <2 | >5 | indicator for eye irritation (only from passive smoking) |
| Nitros-dimethylamine | (1-50) × $10^{-6}$ | — | — | Mutagens under investigation for carcinogenicity |
| $NO_2$ | 0.05-1 | <0.19 | >0.32 | |
| CO | | 2% COHb | 3% COHb | 99.9%[c] |
| | 1-100 | <11 | >30 | Continuous exposure |
| Radon and Progenys | 10-3000 Bq/m³ | -0 | 70 Bq/m³ | Swedish standard for new houses |
| Formaldehyde | 0.05-2 | <0.06 | >0.12 | Long- and short-term |
| $SO_2$ | 0.02-1 | <0.5 | >1.35 | $SO_2$ alone short-term |
| $CO_2$ | 600-9000 | <1800 | >12000 | Japanese standard 1800 mg/m³ |
| $O_3$ | 0.04-0.4 | 0.05 | 0.08 | |
| Asbestos | <10 fibres/m³ | -0 | $10^3$ fibre/m | For long-term exposure |
| Mineral fibres | <10 fibres/m³ | — | — | Skin irritation |
| Organics | | | | |
| Methylene chloride | 0.005-1 | — | 350<br>260 | TLV[d]<br>NIOSH[e] recommendations |
| Trichlormethene | 0.0001-0.02 | — | 270<br>135 | TLV<br>NIOSH recommendations |
| Tetrachloroethene | 0.002-0.05 | — | 335 | TLV |
| 1.4 Dichlorobenzene | 0.005-0.1 | — | 450 | TLV |
| Benzene | 0.01-0.04 | carcinogen | carcinogen | |
| Toluene | 0.015-0.07 | — | 375 | TLV |
| m.p.-Xylene | 0.01-0.05 | — | 435 | TLV |
| n-Nonane | 0.001-0.03 | — | 1050 | ILO (1980) |
| n-Decane | 0.002-0.04 | — | — | |
| Limonene | 0.01-0.1 | — | 560 | TLV turpentine |

[a] All gases were considered on their own without other contaminants.
[b] Typical ranges of concentration given in mg/m³ unless otherwise indicated and for short-term exposures.
[c] According to Environmental Health Criteria No. 4, Geneva World Health Organization, 1977.
[d] TLV (threshold limit values) established by the American Conference of Governmental Industrial Hygenists (19831984). These values are for occupation exposures and should be considered as extreme upper limits for nonoccupational populations for very short-term exposures.
[e] National Institute for Occupational Safety and Health (NIOSH), USA.
[f] International Labor Organization (ILO).
— = no meaningful numbers can be given because of insufficient knowledge.

*From Ref C-24, Table 3.

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C-10. U.S. Consumer Products Safety Commission, Code of Federal Regulations, Title 16, Parts 1303, 1304, 1305 and 1500 (ban of certain commercial practices and hazardous substances regulation).
C-11. U.S. Environmental Protection Agency, Code of Federal Regulation, Title 40, Part 763 (national asbestos regulations).
C-12. U.S. Occupational Safety and Health Administration, Code of Federal Regulations, Title 29, Part 1910 (toxic and hazardous substances).
C-13. U.S. Mine Safety and Health Administration, Code of Federal Regulations, Title 30, Parts 56.5001, 57.5001, 57.5038 and 57.5039 (air quality).
C-14. U.S. Department of Housing and Urban Development, Code of Federal Regulations, Title 24, Part 3280.308 (formaldehyde emission controls for manufactured homes).
C-15. State of Minnesota, Minnesota Laws of 1985, Chapter 216, Section 144.495 (formaldehyde rules for new housing units). 1985.
C-16. U.S. Food and Drug Administration, Code of Federal Regulations, Title 21, Part 801 (maximum acceptable levels of ozone).
C-17. U.S. National Academy of Sciences, Committee on Toxicology, An Assessment of the Health Risks of Seven Pesticides Used in Termite Control (chlordane in military housing), August 1982.
C-18. U.S. National Academy of Sciences, National Research Council, Report of the Panel on Air Quality in Manned Spacecraft of the Committee on Toxicology, *Atmospheric Contaminants in Spacecraft*. June 1972.
C-19. U.S. Naval Research Laboratory, *Navy Submarine Atmospheric Control Manual* (current update of Table 3-7, unclassified defense information).
C-20. American Industrial Hygiene Association, Occupational Exposure and Work Practice Guidelines for Formaldehyde. July 24, 1986.
C-21. U.S. Environmental Protection Agency. *A Citizen's Guide to Radon*. August 1986.
C-22. U.S. Environmental Protection Agency. *Radon Reduction Methods, A Homeowner's Guide*. August 1986.
C-23. Canada Department of National Health and Welfare. *Exposure Guidelines for Residential Indoor Air Quality*. Ottawa. April 1987.
C-24. World Health Organization, Report on a WHO meeting, August 21-24, 1984, *Indoor Air Quality Research*. EURO Reports and Studies 103, Regional Office for Europe, Copenhagen, Denmark.

**This Appendix is not part of this Standard but is included for information purposes only.**

## APPENDIX D
### RATIONALE FOR MINIMUM PHYSIOLOGICAL REQUIREMENTS FOR RESPIRATION AIR BASED ON $CO_2$ CONCENTRATION

Oxygen is necessary for metabolism of food to sustain life. Carbon and hydrogen in foods are oxidized to $CO_2$ and $H_2O$, which are eliminated by the body as waste products. Foods can be classified as carbohydrates, fats, and proteins, and the ratio of carbon to hydrogen in each is somewhat different. The respiratory quotient (RQ) is the volumetric ratio of carbon dioxide produced to oxygen consumed. It varies from 0.71 for a diet of 100% fat to 0.8 for a diet of 100% protein and 1.00 for a diet of 100% carbohydrates (see Ref D-1). A value of RQ = 0.83 applies to a normal diet mix of fat, carbohydrate, and protein.

The rate at which oxygen is consumed and carbon dioxide is generated depends on physical activity. These relationships are shown in Fig. D-2 (see Ref D-2). The breathing rate is shown also. A simple mass balance equation gives the outdoor air flow rate needed to maintain the steady-state $CO_2$ concentration below a given limit.

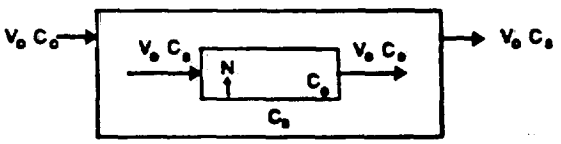


*Fig. D-1. Two Chamber Model*

$$V_o = N/(C_s - C_o) \qquad \text{(D-1)}$$

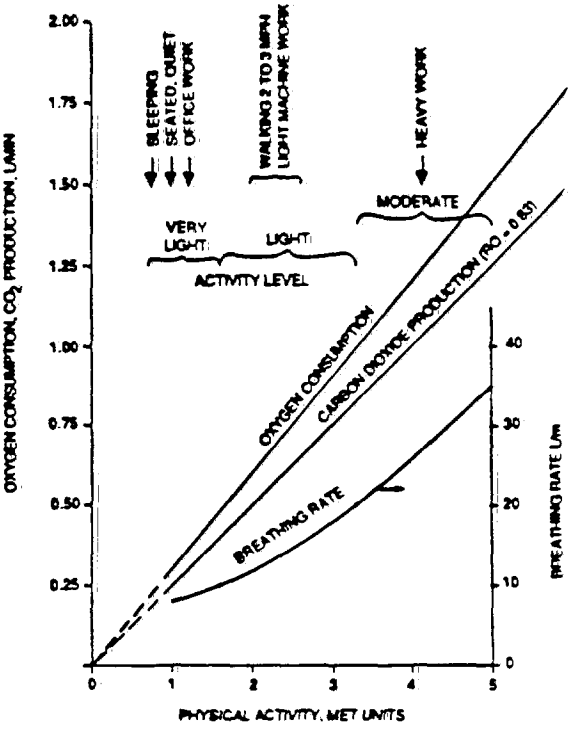


*Fig. D-2 Metabolic Data*

where

$V_o$ = outdoor air flow rate per person
$V_r$ = breathing rate
$N$ = $CO_2$ generation rate per person
$C_r$ = $CO_2$ concentration in exhaled breath
$C_s$ = $CO_2$ concentration in the space
$C_o$ = $CO_2$ concentration in outdoor air

Thus, for an activity level of 1.2 met units (1.0 met = 18.4 Btu/h·ft$^2$), the $CO_2$ generation rate is 0.30 L/min. If the maximum space concentration is to be held to 0.1% and the outdoor concentration is 0.03%, the outdoor air flow needed is,

$$V_o = (0.30)/[(0.001\text{-}0.0003) \times 60]$$
$$= 7.0 \text{ L/s}$$
or 15 cfm per person

Figure D-3 shows the outdoor air flow rate required as a function of physical activity and steady-state room concentration. If the activity level is greater than 1.2 met, the required ventilation must be increased to maintain the same carbon dioxide level.

Also the decrease in oxygen content of the room air can be found from Eq D-1 when oxygen concentration is substituted for carbon dioxide concentration.

$$C_o - C_s = N/V_o \qquad \text{(D-2)}$$

The term $N$ now has a negative value with respect to its use in Eq D-1 since oxygen is consumed rather than generated.

$$C_s = C_o - N/V_o \qquad \text{(D-3)}$$

The oxygen consumption rate is 0.36 L/min when the activity level is 1.2 met. For ventilation at a rate of 15 cfm (429 L/m) and an activity level of 1.2 met units, the room oxygen level will be reduced from an outdoor concentration to 20.9%. Thus the oxygen content of the room is reduced from 21% to 20.9%, a change of only 0.5%. The carbon dioxide is raised from the background of 0.03% to 0.1%, a change of 230%. Thus dilution of carbon dioxide is clearly more significant than replacing oxygen.

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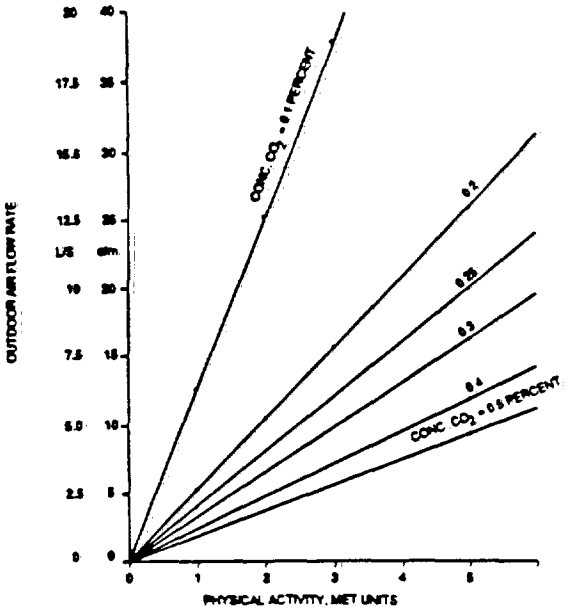


*Fig. D-3 Ventilation Requirements*

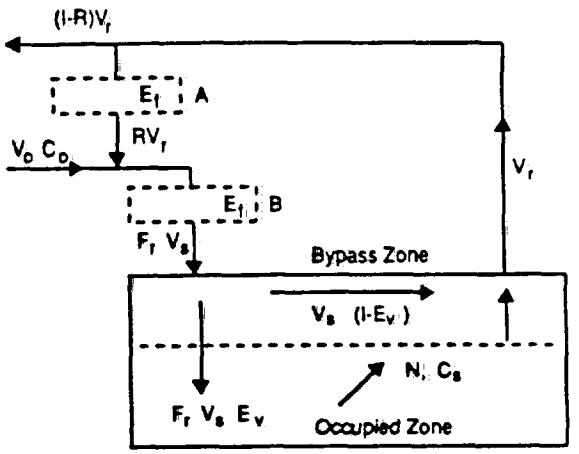


*Fig. E-1 Recirculation and Filtration*

*A, B* = *Filter Location*
*V* = *Volumetric Flow*
*C* = *Contaminate Concentration*
*E* = *Efficiency or Effectiveness*
$F_r$ = *Flow Reduction Factor*
*N* = *Contaminate Generation Rate*
*R* = *Recirculation Flow Factor*

*Subscripts*
*f* = *filter*
*o* = *outdoor*
*r* = *return*
*s* = *supply*
*v* = *ventilation*

## REFERENCES


D-1. McHattie, L.A. *Graphic Visualization of the Relations of Metabolic Fuels: Heat: $O_2$, $CO_2$, $H_2O$: Urine N. J. Applied Physiology*, 15, (4): 677-683. 1960.

D-2. *ASHRAE Handbook—1985 Fundamentals Volume*, Chapter 8. American Society of Heating, Refrigerating, and AirConditioning Engineers, Inc., Atlanta, GA 30329. 1985.


**This Appendix is not part of this Standard but is included for information purposes only).**

## APPENDIX E
## PROCEDURE FOR USE OF CLEANED RECIRCULATED AIR

The amount of outdoor air specified in Table 2 may be reduced by recirculating air from which offending contaminants have been removed or converted to less objectionable forms. Formaldehyde, for example, may be oxidized to water and carbon dioxide. The amount of outdoor air required depends on the contaminant generation in the space, the contaminant concentrations in the indoor and the outdoor air, the filter location, the filter efficiency for the contaminants in question, the ventilation effectiveness, the supply air circulation rate, and the fraction recirculated.

Figure E-1 shows a representative system. A filter may be located in the recirculated airstream (location A) or in the supply (mixed) airstream (location B). The ventilation effectiveness will depend on the location of the supply outlet, the return inlet, and the design and performance of the supply diffuser. Figure E-1 is

**Table E-1 Required Outdoor Air or Space Contaminant Concentration with Recirculation and Filtration**

| Class | Required Recirculation Rate | | | | Required Outdoor Air | Space Contaminant Concentration | Required Recirculation Rate |
|---|---|---|---|---|---|---|---|
| | Filter Location | Flow | Temperature | Outdoor Air | | | |
| I | None | VAV | Constant | 100% | $V_o = \frac{N}{E_v F_r (C_s - C_o)}$ | $C_s = C_o + \frac{N}{E_v F_r V_o}$ | Not applicable |
| II | A | Constant | Variable | Constant | $V_o = \frac{N - E_v RV_r E_f C_s}{E_v (C_s - C_o)}$ | $C_s = \frac{N + E_v V_o C_o}{E_v (V_o + RV_r E_f)}$ | $RV_r = \frac{N + E_v V_o (C_o - C_s)}{E_v E_f C_s}$ |
| III | A | VAV | Constant | Constant | $V_o = \frac{N - E_v F_r RV_r E_f C_s}{E_v (C_s - C_o)}$ | $C_s = \frac{N + E_v V_o C_o}{E_v (V_o + F_r RV_r E_f)}$ | $RV_r = \frac{N + E_v V_o (C_o - C_s)}{E_v F_r E_f C_s}$ |
| IV | A | VAV | Constant | Proportional | $V_o = \frac{N - E_v F_r RV_r E_f C_s}{E_v F_r (C_s - C_o)}$ | $C_s = \frac{N + E_v F_r V_o C_o}{F_r E_v (V_o + RV_r E_f)}$ | $RV_r = \frac{N + E_v F_r V_o (C_o - C_s)}{E_v F_r E_f C_s}$ |
| V | B | Constant | Variable | Constant | $V_o = \frac{N - E_v RV_r E_f C_s}{E_v [C_s - (1-E_f) C_o]}$ | $C_s = \frac{N + E_v V_o (1-E_f) C_o}{E_v (V_o + RV_r E_f)}$ | $RV_r = \frac{N + E_v V_o [(1-E_f) C_o - C_s]}{E_v E_f C_s}$ |
| VI | B | VAV | Constant | Constant | $V_o = \frac{N - E_v F_r RV_r E_f C_s}{E_v [C_s - (1-E_f) C_o]}$ | $C_s = \frac{N + E_v V_o (1-E_f) C_o}{E_v (V_o + F_r RV_r E_f)}$ | $RV_r = \frac{N + E_v V_o [(1-E_f) C_o - C_s]}{E_v F_r E_f C_s}$ |
| VII | B | VAV | Constant | Proportional | $V_o = \frac{N - E_v F_r RV_r E_f C_s}{E_v F_r [C_s - (1-E_f)(C_o)]}$ | $C_s = \frac{N + E_v F_r V_o (1-E_f) C_o}{E_v F_r (V_o + RV_r E_f)}$ | $RV_r = \frac{N + E_v F_r V_o [(1-E_f) C_o - C_s]}{E_v F_r E_f C_s}$ |

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a schematic of a typical system with the supply outlet and the return inlet in the ceiling. It is possible for some supply air to flow directly from the supply to the return, bypassing the occupied zone of the room. This reduces the effectiveness of the ventilation supplied to the space (see Appendix F).

All of the exhaust is shown in Fig. E-1 as being taken from the return airstream. Many systems will have part or all of the exhaust taken directly from the space. If the exhaust air is taken from the ceiling area, it will be subject to the same loss of ventilating efficiency as if it were taken from the return air flow. See Appendix F for its effect on ventilating efficiency, $E_v$. The exhaust air flow, $V_e$, is then equal to all of the exhaust air flow regardless of whether it is taken from the return air or from the room. Then:

$$V_e = V_o \qquad \text{(E-1)}$$

let $V_e'$ be the portion of air exhausted form the return duct, and $V_e''$ be the portion exhausted directly from the room. Then:

$$V_e = V_e' + V_e'' \qquad \text{(E-2)}$$

and

$$V_r = V_i - V_e'' \qquad \text{(E-3)}$$

The material balance at the supply outlet is:

$$V_s = V_o + RV_r = (V_e' + V_e'') + RV_r \qquad \text{(E-4)}$$

$$RV_r = V_s - (V_e' + V_e'') \qquad \text{(E-5)}$$

$RV_r$ is the recirculated air and $V_r$ is the return air. Therefore:

$$R = [V_i - V_e' + V_e'')]/V_r \qquad \text{(E-6)}$$

Variable-air-volume (VAV) systems reduce the circulation rate when the thermal load is satisfied. This is accounted for by a flow reduction factor $Fr$. The supply air temperature is normally held constant in a VAV system. Constant-volume systems require a variable supply air temperature. VAV systems also may have a constant or proportional outdoor air flow rate.

A mass balance for the contaminant may be written to determine the space contaminant concentration for each of the system arrangements. The various permutations for the air-handling and distribution systems are described in Table E-1. There are seven variations. The mass balance equations for computing the space contaminant concentration for each system are presented in Table E-1.

If the allowable space contamination is specified, the equations in Table E-1 may be solved for the outdoor flow rate $V_o$. When the outdoor air flow rate is specified, the equations may be solved for the resulting contaminant concentration as shown in Table E-1.

Filters are effective for removing particles. They are less effective or ineffective in removing gases and vapors. Therefore, when designing a filtration system, consideration must be given to contaminants that are poorly filtered or not filtered at all. The ventilating rate may only be reduced until some contaminant reaches its maximum acceptable limit.

**This Appendix is not part of this Standard but is included for information purposes only.**

## APPENDIX F
### VENTILATION EFFECTIVENESS

**Stratification Model.** A model for ventilation effectiveness can be derived by considering a typical HVAC air-handling system as shown schematically in Fig. F-1. It is possible that a fraction, $S$, of the supply air may bypass directly to the return inlet without mixing at the occupied level; i.e., below the dotted line in Fig. F-1.

The total outdoor air in the supply air is $V_{oa}$.

The fraction of the supply air that stratifies and bypasses directly to the return is designated by $S$. This model applies to the forced ventilation system and excludes the effects of passive infiltration. The amount of outdoor air supplied to the space is:

$$V_{oa} = V_o + R \times S \times V_{oa} \qquad \text{(F-1)}$$

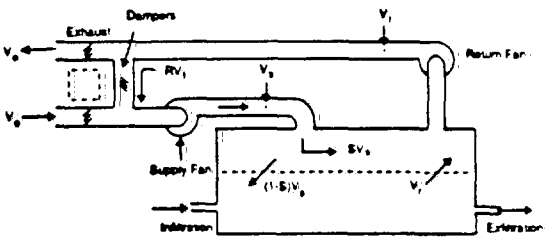


*Fig. F-1. Typical Air Distribution System*

The amount of unvitiated (unused) air that is exhausted is:

$$V_{ue} = (1 - R)S\,V_{oa} \qquad \text{(F-2)}$$

The ventilation efficiency then can be defined as:

$$E_v = [V_o - V_{ue}]/V_o \qquad \text{(F-3)}$$

Combining equations F-1, F-2, and F-3,

$$E_v = [1 - S]/[1 - RS] \qquad \text{(F-4)}$$

Equation F-4 defines the effectiveness with which the outdoor air is circulated to the occupied space in terms of a stratification or mixing factor $S$ and the recirculation factor $R$. If there is no exhaust flow, $R = 1$ and the effectiveness = 100%. If, however, there is both stratified flow and recirculation, outdoor air can pass through the system without ever being used to dilute contaminants at the occupied level. This ventilation loss also represents an energy loss.

### REFERENCE

F-1. Janssen, J.E. *Ventilation Stratification and Air Mixing, Indoor Air*, Vol. 5. Proc. of the 3rd Intern. Conference on Indoor Air Quality and Climate, Stockholm, Sweden, 1984. Sponsored by USEPA, ASHRAE, GRI, EPRI and European organizations. 1984.

**This Appendix is not part of this Standard but is included for information purposes only.**

## APPENDIX G
### RATIONALE FOR LAG OR LEAD TIME FOR TRANSIENT OCCUPANCY

When spaces such as classrooms, auditoriums, or offices are unoccupied for several hours and then occupied, operation of the ventilation system may be delayed to use the capacity of the air in the space to dilute contaminants. This applies to cases where the inside contaminants are associated only with human occupancy and where contaminants are dissipated by natural means during long vacant periods. The operation of the ventilation system can then be delayed until the concentration of contaminants reaches the acceptable limit associated with the minimum ventilation requirements at steady state.

The concentration of any contaminant $C$ in the absence of ventilation in a given space of volume $v$ is expressed as follows:

$$C_t = \frac{N \cdot t}{v} \qquad \text{(G-1)}$$

where $N$ is the contaminant generation rate and $t$ is time. The contaminant concentration $C_s$ under a steady-state condition with ventilation rate $V$ is:

$$C_s = \frac{N}{V} \qquad \text{(G-2)}$$

The maximum permissible ventilation delay time after the space is occupied is when $C_t$ equals $C_s$, or:

$$t = \frac{v}{V} \qquad \text{(G-3)}$$

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This equation is plotted in Fig. 4 for various ventilation rates in cfm/person (L/s per person) and space volume in $ft^3$/person (L/person). When contaminants are generated independent of people or their activities, and the contaminants do not present a short-term health hazard, ventilation may be shut off during unoccupied periods. In these cases, however, ventilation must be provided in advance of the time of occupancy, so that acceptable conditions will exist for people at the start of occupancy. It is impractical to operate the ventilation system at the minimum requirement until steady state is reached, because this is approached asymptotically with time and may take several hours to reach practical equilibrium. An engineering estimate of a permissible contaminant level of 1.25 times the steady-state value has, therefore, been selected as the maximum level at the time of occupancy. The occupants would, for a time, be subjected to somewhat higher contaminant concentrations than the steady-state value. It is postulated that the factor of safety implicit in the concentrations in 6.1.3 are adequate so that, for practical purposes, the required air quality is provided over the entire occupancy period.

When an initially contaminated room with a level of concentration $C_i$ is diluted by a given rate of ventilation $V$, the time required to lower the concentration to a fraction $X$ above the final steady-state concentration level can be expressed as follows:

$$t = (v/V)\ln\left[\frac{(C_i)\,V/N - 1}{X}\right] \qquad \text{(G-4)}$$

where:

$t$ = time
$v$ = room volume
$V$ = ventilation rate
$N$ = contaminant generation rate
$C_i$ = initial concentration

Figure 5 is a plot of this relationship, where $C_i$ is assumed to be approximately 10 times the steady-state value and $X$ = 0.25 or 25%.

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## APPENDIX H
## RATIONALE FOR REDUCING OUTDOOR AIR WHEN LOADS ON A MULTI-ZONE SYSTEM ARE UNEQUAL

Building HVAC systems often serve more than one room or zone. When the ventilating requirements for different rooms or zones are unequal, some of the return air is recirculated, and exhaust is taken from the return air, it is possible to reduce the fraction of outdoor air in the total supply below that required for the critical space, i.e., the space with the greatest ventilation requirement. Derivation of the formula that specifies the fraction of the outdoor air required in the total supply is as follows:

A two-chamber model may be used as shown in Fig. H-1. One chamber, represented by subscript $c$, represents the critical zone or zone with the highest ventilation load. The other chamber represents the sum of the other zones or rooms that require a smaller fraction of outdoor air in their supplies than the critical zone.

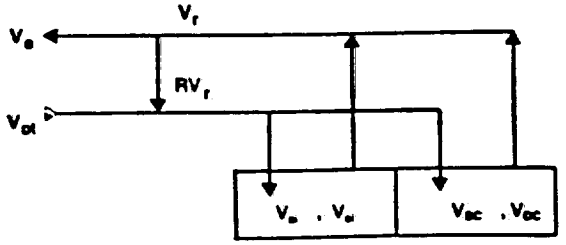


*Fig. H-1. Multi-Zone Model*

$V_e$ = exhaust from the system
$V_{ot}$ = outdoor air supply corrected to account for recirculation
$V_{st}$ = total supply air flow
$V_r$ = return air flow
$V_{oi}$ = outdoor air flow supplied to zone i
$V_{on}$ = sum of the outdoor air supplied to all zones

$$V_{on} = \sum_{i=1}^{n} V_{oi}$$

$V_{si}$ = supply air flow to zone i
$V_{sn}$ = sum of supply flows to all zones

$$V_{sn} = \sum_{i=1}^{n} V_{si}$$

$V_{oc}$ = outdoor air supplied to critical zone
$V_{sc}$ = supply air flow to critical zone
$F$ = fraction of outdoor air in supply to critical zone

$$F = V_{oc}/V_{sc} \qquad \text{(H-1)}$$

$R$ = fraction of return air that is recirculated, i.e.,

$$R = [(V_r - V_e)/V_r] = [V_{st} - V_{ot})/V_{st}] \qquad \text{(H-2)}$$

Note that

$$V_{on} = \sum_{i=1}^{n} V_{oi} \qquad \text{(H-3)}$$

and

$$V_{sn} = \sum_{i=1}^{n} V_{si} \qquad \text{(H-4)}$$

Then, by definition for the critical zone,

$$V_{oc}/V_{sc} \geq V_{on}/V_{sn} \qquad \text{(H-5)}$$

Thus, if the supply contains a fraction of outdoor air needed to satisfy the critical zone, the other zones will be overventilated and their return will contain unvitiated or "unused" outdoor air. A fraction, $R$, of the return air then can be recirculated to supply some of the outdoor air needed by the critical zone. This will reduce the amount of outdoor air needed by the system.

If $F$ is the fraction of outdoor air in the supply to the critical zone, the flow rate of unvitiated (unused) outdoor air in the return from the overventilated zones is,

$$FV_{st} - V_{on} \qquad \text{(H-6)}$$

The fraction of this unvitiated return air that is recirculated is

$$R[FV_{st} - V_{on}] \qquad \text{(H-7)}$$

The total amount of useable outdoor air, $V_{st}$, in the supply then is,

$$V_{ot} + R\,[FV_{st} - V_{on}] \qquad \text{(H-8)}$$

The ventilation requirement of the critical space is met when the supply air, $V_{st}$, contains a fraction of outdoor air equal to,

$$FV_{st} = V_{ot} + R[FV_{st} - V_{on}] \qquad \text{(H-9)}$$

From the definitions of the recirculation fraction, $R$, given by Eq H-2,

$$FV_{st} = V_{ot} + [(V_{st} - V_{ot})/V_{st}][FV_{st} - V_{on}] \qquad \text{(H-10)}$$

Equation H-10 can now be solved for the total outdoor air, or fresh air needed to satisfy all zones.

$$FV_{st} = V_{ot} + FV_{st} - FV_{ot} - V_{on} + V_{ot}V_{on}/V_{st} \qquad \text{(H-11)}$$

$$O = V_{on}[1 - F + V_{on}/V_{st}] - V_{on} \qquad \text{(H-12)}$$

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Therefore,

$$V_{ot} = V_{on}/[1 + V_{on}/V_{st} - F] \quad \text{(H-13)}$$

or substituting the value for $F$ and dividing both sides of the equation by the total supply $V_{st}$:

$$V_{ot}/V_{st} = [V_{on}/V_{st}]/[1 + ([V_{on}/V_{st}) - (V_{oc}/V_{sc})] \quad \text{(H-14)}$$

Equation H-14 can be written

$$Y = X/(1 + X - Z) \quad \text{(H-15)}$$

where

$Y = V_{ot}/V_{st}$, the corrected fraction of outdoor air in the total supply

$X = V_{on}/V_{st}$, the uncorrected fraction of outdoor air in the total supply

$Z = F$, the fraction of outdoor air in the supply of the critical zone.

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## POLICY STATEMENT DEFINING ASHRAE'S CONCERN FOR THE ENVIRONMENTAL IMPACT OF ITS ACTIVITIES

ASHRAE is concerned with the impact of its members' activities on both the indoor and outdoor environment. ASHRAE's members will strive to minimize any possible deleterious effects on the indoor and outdoor environment of the systems and components in their responsibility, while maximizing the beneficial effects which these systems provide, consistent with the accepted standards and the practical state of the art.

ASHRAE's short-range goal is to ensure that the systems and components within its scope do not impact the indoor or outdoor environment to a greater extent than specified by the standards as established by itself and other responsible bodies.

As an on-going goal, ASHRAE will, through its Standards Committee and extensive technical committee structure, continue to generate up-to-date standards where appropriate and adopt, recommend and promote those new and revised standards developed by other responsible organizations.

Through its Handbook, appropriate chapters will contain up-to-date standards and design considerations as the material is systematically revised.

ASHRAE will take the lead with research and dissemination of environmental information of its primary interest and will seek out and disseminate information from other responsible organizations which is pertinent, as guides to updating standards.

The effects of the design and selection of equipment and systems will be considered within the scope of the system's intended use, and expected misuse. The disposal of hazardous materials, if any, will also be considered.

ASHRAE's primary concern for environmental impact will be at the site where equipment within ASHRAE's scope operates. However, energy source selection and the possible environmental impact due to the energy source and energy transportation will be considered where possible. Recommendations concerning energy source selection should be made by its members.

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# A COMPUTER SIMULATION OF INDOOR AIR QUALITY WHICH MODELS CHANGES IN POINT SOURCES AND VENTILATION

Joe F. Pedelty and Larry C. Holcomb*

Holcomb Environmental Services, 17375 Garfield Road, Olivet, MI 49076, U.S.A.

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## ABSTRACT

The subject of indoor air quality is generating increasing concern in the public and political arena. In spite of its publicity it is still a fairly technical subject, the health effects of which are poorly understood. A model is described which will visually illustrate the indoor air quality in a specific situation, the parameters which affect it and how it compares with occupational health standards. Environmental tobacco smoke is evaluated as one point source which can affect indoor air quality.

## INTRODUCTION

The subject of indoor air quality is one of continually increasing concern to the public. More and more information is available regarding the nature and types of chemicals found in indoor environments and people are demanding access to that information. Unfortunately the subject of indoor air quality is a fairly technical one and not well understood by those most affected by it. Terms such as parts per billion and micrograms per cubic meter often mean little if anything to the public. The toxicological implications of the chemicals measured at these concentrations are still open to debate among the experts and, as such, cannot be easily boiled down into compact ideas for general consumption.

In spite of, or perhaps because of, the problems in accurately disseminating this information in a non-technical manner, special interest groups have a tendency to take one small portion of the available information and create a series of "sound bite" releases of it to further their own cause. This does nothing towards helping the public become more understanding of the causes or overall risks of indoor air pollution. On the contrary, it tends to foster either an unreasonable phobia or an equally unreasonable indifference to the situation.

Because of the technical nature of the subject and the misconceptions that are being fostered at some levels, there is a definite need for visual aids which will simply, yet accurately, demonstrate indoor air quality as it exists in typical environments and the factors which can affect it.

This paper describes a computer model which will generate charts on which concentrations of various chemicals typically found in indoor air are represented by a specific number of dots. It will allow people to view various levels of indoor air components in relation to each other and in relation to a recognized set of health standards, the OSHA Permissible Exposure Limits (PEL). The computer program is set up so that the various factors affecting these levels in indoor environments can be manipulated to show the effects of improving ventilation and/or removing individual point sources by simply changing the values entered into the program.



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Literature data available on general indoor air quality and point sources which have been shown to affect it were used to test the model. The point source which was chosen to test the model was environmental tobacco smoke (ETS). ETS is the only visible constituent of indoor air and it has a very distinctive odor. As a result, it is usually accused of being the major source of chemicals in the air. It has also become the subject of a considerable amount of controversy and research. This makes it an attractive point source for demonstration purposes.

It is important to keep in mind that all of the data used to test the model were taken from existing studies on indoor air quality. The level of constituents used in the model and the percent assigned as being produced by ETS are only as accurate as the information available in the literature. More refined studies such as those by Oldaker (1) will allow the model to predict more accurately the true relationship between constituents dependent upon source strength, ventilation and filtration.

## CHART DEVELOPMENT

The goal of this project is to be able to represent various levels of air quality visually so that they can be easily interpreted by non-technical persons. To do this, a computer model was developed which would represent the concentrations of up to twenty five different components of indoor air with a given number of dots on a chart. It is important to be able to produce an accurate representation of these substances on the charts so that the person using them is given a relatively unbiased view of the constituents in a room and the concentrations produced by the individual source being evaluated.

One of the important stages in producing an accurate chart is determining what concentration of a component should be represented as a unit (one dot on the chart), the "unit concentration". Since the primary concern related to indoor air quality is that of health effects, the unit concentrations are based on OSHA's Permissible Exposure Limits (PEL) using their 8 hour Time Weighted Average (TWA) concentrations. OSHA, over the years, has attempted to regulate substances in the air in workplace environments at levels where, below which, they calculated that there was an insignificant chance for a health risk to the workforce.

Our unit concentrations and dot calculations were developed as follows:

1. The level at which OSHA regulates the individual component was divided by 500. This number was chosen because, using the concentrations of chemicals commonly found in indoor environments, it produces a number of dots on the charts which are easily managed. (OSHA regulates carbon monoxide at 35 ppm. 35 ppm divided by 500 = 0.07 ppm. The unit concentration for carbon monoxide is 0.07 ppm.)
2. The concentration of the component is then divided by the "unit concentration". (e.g. Carbon monoxide levels in indoor environments may be found to be 3.0 ppm. 3.0 ppm divided by 0.07 ppm = 42.8. Carbon monoxide would be represented on the chart with 43 dots.)

In using this procedure, the chemicals which are regulated by OSHA at lower levels would have a smaller unit concentration. They would then be given more dots on the chart relative to those regulated by OSHA at a higher concentration.

For example: Ammonia is regulated by OSHA at 50 ppm. Sulfur dioxide is regulated by OSHA at 2 ppm. The unit concentration for ammonia would be 0.1 ppm. The unit concentration for sulfur dioxide would be 0.004 ppm. If both gases were present in a room at a concentration of 1 ppm, ammonia would be represented by 10 dots on the chart and sulfur dioxide would be represented by 250 dots on the chart.

Thus, indoor air components are shown on the charts relative to their contribution to an established exposure limit and not just relative to their concentration in the room.

Chart Descriptions: To effectively demonstrate indoor air components in a specific environment, the effect of point source modifications, ventilation modifications and a toxicological assessment of them, five different charts are created.

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Chart one shows a mixture of the chemical components chosen to be modeled that are present in concentrations which add up to 100% of the OSHA PEL. OSHA regulates mixtures of chemicals by determining the concentration of a given chemical in the air and dividing that concentration into the PEL for that chemical. This produces a figure which represents the percentage of the PEL taken up by that chemical. This process is repeated for each of the individual components, generating percentage values for each component. These percentages are then added together. If the sum of the values is less than 1.0 (or 100%) then the PEL for the mixture has not been reached. If the total of the percentages is equal to 1.0 then the concentration of the mixture is at the PEL for that mixture. The first chart is generated by performing these calculations and determining what concentrations of the chosen chemicals will combine to total 100% of the PEL for that mixture. Each of these calculated concentrations is then assigned a number of dots on the chart using the "unit concentration" for that chemical.

Since the "unit concentration" for any given chemical is 1/500 of the OSHA PEL of that chemical, this mixture at the OSHA PEL has 500 dots on it. This first chart provides a visual representation of an environment which is believed by OSHA to provide no significant health risk if one is exposed to it 8 hrs/day, 5 days/week. This can provide the basis for a semi-quantitative comparison of any given indoor environment with a mixture of the same substances that is at the OSHA PEL.

Chart two shows a mixture of the same chosen indoor air components in concentrations at which they are measured in the environment being monitored.

Chart three shows the result of simply removing the point source. To do this, two sets of values are used; the measured concentrations used for chart two and the calculated percentage of each component produced by the point source being monitored. Entering these two values into the computer model generates a chart which shows the effects of simply removing the point source.

Chart four illustrates the effect of improved ventilation on the indoor air illustrated in chart two.

The model that was used to predict the new indoor component concentrations that would be found with improved ventilation is dependent upon three variables: A) the outdoor concentration of the component, B) the rate of the production of the component indoors and C) the ventilation of the building.

A) The outdoor contribution of each component can be determined by measuring the same chosen components outside as well as inside of the building.

B) The rate of production of the various components indoors is calculated using data known to apply to the source being evaluated or by using the following equation: $S = A*V*(Cin - Cout)$.

Where: S = Source Strength
A = Current Ventilation Rate
V = Room Volume
Cin and Cout = Indoor and outdoor concentration of the chemical.

C) The ventilation rate is the desired improved rate.

The following formula from Wadden and Scheff (2) was used to model the effects of ventilation rates on indoor air concentrations:

$$Ci=\frac{K[(Q(1-F)(Co))+S)][1-2.7183^{-K(Q+Q1F)T}]+[Cs(2.7183^{-K(Q+Q1F)T}]}{K(Q+Q1F)}$$

Where Ci = The new indoor concentration.
Co = The outdoor contribution to the indoor conc.
Cs = The starting indoor concentration.
Q = The make-up ventilation rate in air changes per hour (ach).
Q1 = The recirculation ventilation rate in ach.
S = The net source emission rate.

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Source: https://www.industrydocuments.ucsf.edu/docs/zqyx0000

T = The time at the new ventilation rate.
K = Mixing factor.
F = Filter efficiency.

Entering the value for these variables into the computer model generates a chart representing the chosen components in the concentrations they would have at that ventilation rate.

Chart five will calculate the effect of removing the point source and improving ventilation. It combines the calculations used to generate charts 3 and 4 to generate a new chart with both modifications in place.

## MODEL TEST USING LITERATURE DATA

To test this model the available literature on indoor air quality and ETS was reviewed. Though the program will evaluate up to twenty five different components, thirteen components which are commonly found in indoor air were chosen to be evaluated. These components were chosen because they are commonly found in indoor environments and there is a substantial amount of literature available pertaining to them. Levels of these components which had been measured in indoor environments, and the percentage of the chemicals believed to be produced by ETS were recorded. Table 1 shows the results of this literature search.

TABLE 1: INDOOR AIR CONSTITUENTS: CONCENTRATION RANGES FROM THE LITERATURE

| COMPONENT | RANGE CITED | CITATION FOR INDOOR AIR | % FROM ETS | CITATION FOR % FROM ETS |
|---|---|---|---|---|
| Carbon Monoxide | ND - 242 ppm | 3, 4, 5, 6, 7 8, 9, 10, 11 | 15% | 9, 10 |
| Ammonia | 5 ppm | 12 | 3% | 5 |
| Formaldehyde | 0.02 - 3.6 ppm | 8, 12, 13, 14, 15, | 5% | 7, 16 |
| Ozone | 0.03 - 0.23 ppm | 17 | 0% | |
| Oxides of Nitrogen | ND - 800 ug/m3 | 5, 7, 8, 11 12, 13, 14, 18 | 12% | 7, 11, 18 |
| RSP | 0 - 1,088 ug/m3 | 1, 5, 7, 9, 10, 11, 13, 14, | 50% | 19 |
| Alkanes | 34 - 327 ug/m3 | 20 | 5% | 21 |
| Chlor. Hyd. Car. | 2.5 - 43 ug/m3 | 19, 20, 21 | 0% | |
| Benzene | ND - 109 ug/m3 | 4, 7, 11, 14, 21 | 35% | 21 |
| Sulfur Dioxide | ND - 0.17 ppm | 8, 13 | 20% | 22, 11 |
| Nicotine | ND - 112.4 ug/m3 | 1, 3, 7, 9, 10, 11, 22, 23, 24, 25, 26, 27 | 100% | 5 |
| Benzo(a)Pyrene | 0.25 - 23.4 ng/m3 | 7, 11, 14 | 5% | 7, 11 |
| Biologicals | 500 - 1,000 cfu/m3 | 14 | 0% | |

The second part of this project involved the estimation of a typical concentration for each chemical. This was, by necessity, a very subjective task. The concentrations of a given component documented in indoor environments often vary by more than an order of magnitude. In some of the studies, concentrations measured in rooms with the point source (ETS) present showed lower levels of constituents than rooms with the source absent. It was necessary, though, to be able to assign specific values based on the current literature that may be expected to be found in typical indoor environments. To assist with the selection process, the following assumptions were built into the process:

1. The ventilation in the building (make up air) is less than 5 cfm/person.
2. The building is relatively new. Hence it is a "tight" building and the furnishings and building materials are still emitting some of the compounds used in their manufacture.
3. Approximately 20% of the people in the building are smokers.
4. Smokers are estimated to smoke one cigarette/hr/smoker.
5. Occupancy of the building is assumed to average 1 person/150 sq.ft.

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6. The ceilings in the building are 8 ft. high.
7. The building is in an urban setting. Automotive exhaust fumes and ozone are significant sources of chemicals in the outdoor air.
The results of these value assignments are shown in table 2.

TABLE 2: TYPICAL INDOOR AIR COMPONENTS

| COMPONENT | TYPICAL CONCENTRATION | CONCENTRATION LESS ETS |
|---|---|---|
| Carbon Monoxide | 3.5 mg/m3 (3 ppm) | 3.0 mg/m3 (2.6 ppm) |
| Ammonia | 2.0 mg/m3 (3 ppm) | 1.94 mg/m3 (2.9 ppm) |
| Formaldehyde | 600 ug/m3 (300 ppb) | 570 ug/m3 (214 ppb) |
| Ozone | 40 ug/m3 (20 ppb) | 40 ug/m3 (20 ppb) |
| Oxides of Nitrogen | 100 ug/m3 (85 ppb) | 88 ug/m3 (75 ppb) |
| RSP | 80 ug/m3 | 40 ug/m3 |
| Alkanes | 50 ug/m3 (17 ppb) | 47.5 ug/m3 (16 ppb) |
| Chlor. Hyd. Car. | 30 ug/m3 (5.5 ppb) | 30 ug/m3 (5.5 ppb) |
| Benzene | 15 ug/m3 (5 ppb) | 9.8 ug/m3 (3.25 ppb) |
| Sulfur Dioxide | 15 ug/m3 (6 ppb) | 12 ug/m3 (4.8 ppb) |
| Nicotine | 8 ug/m3 (1.2 ppb) | 0.0 ug/m3 (0.0 ppb) |
| Benzo(a)Pyrene | 4 ng/m3 (0.36 ppt) | 3.8 ng/m3 (0.34 ppt) |
| Biologicals | 500 cfu/m3 | 500 cfu/m3 |

The OSHA PEL for each of the thirteen components chosen was needed to determine the "unit concentration" for each component. Table 3 shows the OSHA PEL for each of the thirteen components chosen to test the model (28). Some of the components did not have a specific PEL associated with them.

TABLE 3: OSHA PERMISSIBLE EXPOSURE LIMITS

| Component | OSHA PEL |
|---|---|
| Carbon Monoxide | 35 ppm |
| Ammonia | 50 ppm |
| Formaldehyde | 3.0 ppm |
| Ozone | 0.1 ppm |
| Oxides of Nitrogen* | 5.0 ppm |
| RSP | 5 mg/m3 |
| Alkanes* | 50 ppm |
| Chlor. Hyd. Car.* | 10 ppm |
| Benzene | 1 ppm |
| Sulfur Dioxide | 2 ppm |
| Nicotine | 0.07 ppm |
| Benzo(a)Pyrene | 0.02 ppm |
| Biologicals | 50 cfu/m3 |

*These components are mixtures of chemicals. A value for the PEL was chosen which is believed to be a conservative representation of the components of that mixture. Since biologicals are not regulated, a value was chosen which allowed that component to be represented at a level similar to the rest.

The outdoor contribution of each component is given in Table 4. These values were estimated from papers by Sterling (8) and Yocum (17). These papers listed various constituents commonly measured in indoor environments and the relative contribution of outdoor and indoor sources of each. They are also based on the assumption that the indoor source is in an urban area where automotive exhaust and ozone are significant sources of chemicals.

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TABLE 4: PERCENTAGE OF INDOOR AIR CONSTITUENTS PRODUCED FROM OUTDOOR SOURCES

| Component | % From Outdoor Sources |
|---|---|
| Carbon Monoxide | 60% |
| Ammonia | 0% |
| Formaldehyde | 0% |
| Ozone | 100% |
| NOx | 20% |
| RSP | 25% |
| Alkanes | 60% |
| Chlor. H.C. | 30% |
| Benzene | 50% |
| SO2 | 60% |
| Nicotine | 0% |
| BaP | 80% |
| Biologicals | 60% |

The rate of production of the various components was calculated using the following steps.

1. The production of various components found in ETS (in ug/cigarette) was calculated using the steps and assumptions outlined below.

A. The Mainstream Smoke (MS) Quantities and SS/MS Ratios were taken from the tables in the Surgeon General's and NAS reports on ETS.

B. The SS Qty./cigarette was determined by calculating the value that is 1/3 the way between the min. and max. value of each category. This is an estimate of the mean value based on Oldaker (1) who states that individual values tend to be distributed log-normally throughout the range.

C. These "average" MS quantities and "average" SS/MS ratios were then multiplied together to provide "average" quantities of each component generated in the SS portion of the cigarette smoke.

D. * Indicates components for which no figures are cited in the NAS or Surgeon General's reports or for which direct measurements exist in other publications.

E. RSP values are calculated as the midpoint between the figure of 26 mg/cigarette cited in the NAS report for calculating RSP concentrations based on the number of cigarettes smoked in a room and a measurement of 10 mg/cigarette particulate production in SS by Lofroth (6).

F. Formaldehyde values are from a paper by Godish (16) which assessed the contribution of ETS to formaldehyde concentrations in indoor air.

These values can be seen in Table 5.

TABLE 5: SIDESTREAM (SS) PRODUCTION QUANTITIES FROM CIGARETTE SMOKE

| Component | MS Qty. (ug) Min. | Max. | SS/MS Ratio Min. | Max. | SS Qty./cigarette (ug) |
|---|---|---|---|---|---|
| Carbon Monoxide | 10000 | 23000 | 2.5 | 4.7 | 46344 |
| Ammonia | 50 | 130 | 40 | 170 | 6389 |
| Formaldehyde | * | * | * | * | 740 |
| Ozone | 0 | 0 | 0 | 0 | 0 |
| NOx | 100 | 600 | 4 | 10 | 1600 |
| RSP | * | * | * | * | 18000 |
| Alkanes | * | * | * | * | |
| Chlor. H.C. | 0 | 0 | 0 | 0 | 0 |
| Benzene | 12 | 48 | 5 | 10 | 160 |
| SO2 | * | * | * | * | |
| Nicotine | 1000 | 2500 | 2.6 | 3.5 | 4250 |
| BaP | 20(ng) | 40(ng) | 2.5(ng) | 3.5(ng) | 75.5(ng) |
| Biologicals | 0 | 0 | 0 | 0 | 0 |

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2. These were combined with the occupancy, room size and smoking rate assumptions to generate point source strengths in ug/m3/hr or ppb/hr. as is shown in Table 6 using the following information.

A. At an assumed density of 1 person/150 ft2 and 8 ft. ceiling there will be 1 person/1200 ft3.

B. If 20% of the people are smokers there will be 1 smoker/6000 ft3.

C. At 35.32 ft3/1 m3 there will be 1 smoker/170 m3.

D. If smokers smoke 1 cigarette/hr then the quantity of a given component produced by one cigarette will be released into 170 M3 of volume every hour. Thus, 1 cigarettes qty/170 = ug/m3/hr of production of that component.

E. * Alkane and sulfur dioxide source strengths are simply estimates based on the values used to generate charts 1 & 2.

F. Literature sources indicate that some components of ETS are accumulated in a room at much lower rates than would be calculated from the sidestream generation rates. This is due to oxidation reactions, evaporation and deposition to various surfaces in the room. These "sinks" cause the removal of these components which is independent of ventilation. Based on the figures cited in Baker et al. (29) the following factors were used to determine net source strengths (production rate minus removal by sinks) of the following components: A) Oxides of Nitrogen, Source strength X 0.8 = Net Source Strength; B) RSP, Source strength X 0.4 = Net Source Strength; C) Alkanes, Source strength X 0.8 = Net Source Strength; D) Nicotine, Source strength X 0.5 = Net Source Strength.

TABLE 6: SOURCE STRENGTHS FOR VARIOUS COMPONENTS OF ETS

| Component | Source Strength (ug/m3/hr) | (ppb/hr) | Net Source Strength (ppb/hr) |
|---|---|---|---|
| Carbon Monoxide | 272 | 234 | |
| Ammonia | 38 | 54 | |
| Formaldehyde | 4.4 | 2.8 | |
| Ozone | 0 | 0 | |
| Oxides of Nitrogen | 9.4 | 8 | 6.4 |
| RSP | 106 | | 42.4 (ug/m3) |
| Alkanes | * | 0.5 | 0.4 |
| Chlor. Hyd. Car. | 0 | 0 | |
| Benzene | 0.9 | 0.3 | |
| Sulfur Dioxide | * | 1.0 | |
| Nicotine | 25 | 3.8 | 1.9 |
| Benzo(a)Pyrene | 0.44(ng) | 0.04 (ppt/hr) | |
| Biologicals | 0 | 0 | |

Though it is assumed that there are other indoor sources for the components, the production rate is calculated to be from the smokers only.

To show the result of removing the point source (ETS), we used two sets of values; the typical concentrations used in table two and the calculated percentage of each component produced by ETS depicted in table 1. Entering these two values into the computer model generates a chart which shows the effects of simply removing the point source, in this case, removing environmental tobacco smoke.

To illustrate the effect of improved ventilation on the indoor air illustrated in chart two improved ventilation rates needed to be assigned. The ventilation rate was increased to 20 cfm/person, the rate recommended by the American Society of Heating, Refrigeration and Air Conditioning Engineers (ASHRAE). With the assumptions of 1 person/150 ft2 and 8 ft. ceilings this is the equivalent of 1 air exchange/hr (1 ach). The mixing factor and time at ventilation were assigned values of 1.0 and 24 hours, respectively.

The purpose of this overall exercise was to generate visual representations of indoor air with and without a specific point source (in this case, ETS) and with and without

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improved ventilation/filtration. This is made possible once one determines:

a. The typical concentrations of the components to be evaluated.
b. The point source of concern.
c. The rate of production of the components in an indoor environment.
d. The percentage of each component produced from outside sources.
e. Desired ventilation parameters.

It was also possible to compare these levels of indoor air quality with a similar indoor environment that would be the maximum acceptable concentration of components allowed by OSHA. The results of this can be seen in charts 1 - 5.

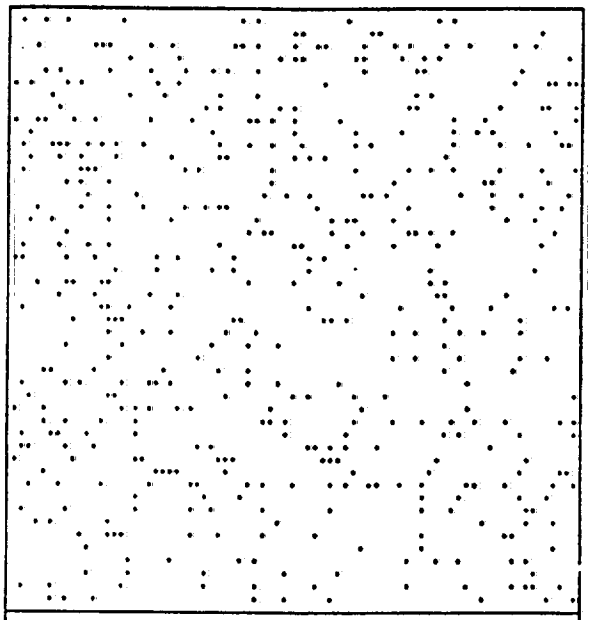

CHART #1
INDOOR AIR CONSTITUENTS AT THE OSHA PEL

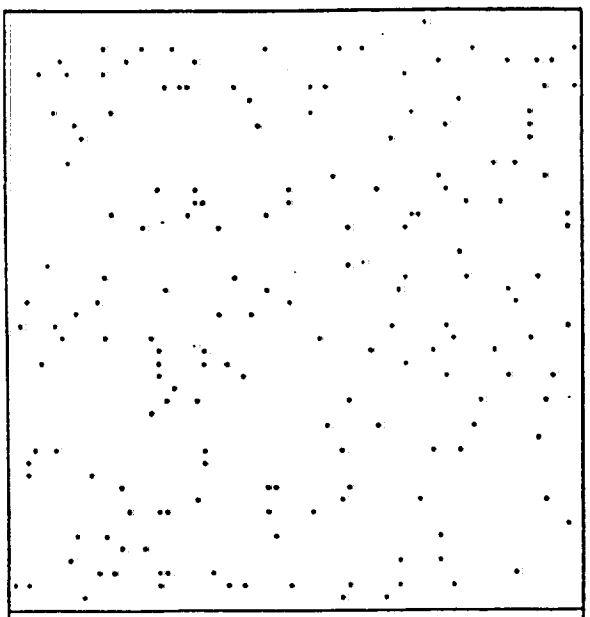

CHART #2
TYPICAL INDOOR AIR WITH SMOKING ALLOWED

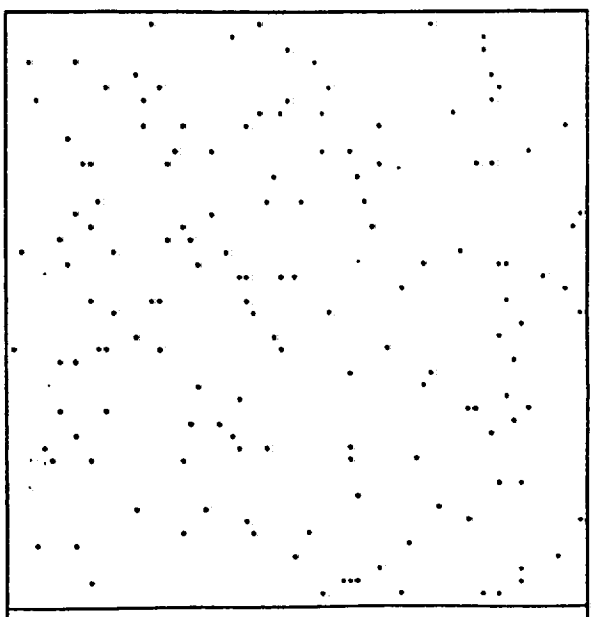

CHART #3
TYPICAL INDOOR AIR WITH NO SMOKING

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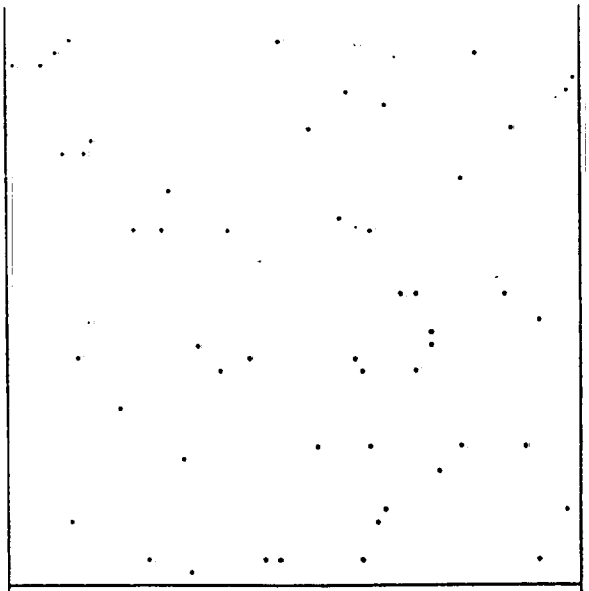

CHART #4
TYPICAL INDOOR AIR WITH VENTILATION INCREASED TO 20 CFM/PERSON AND SMOKING ALLOWED

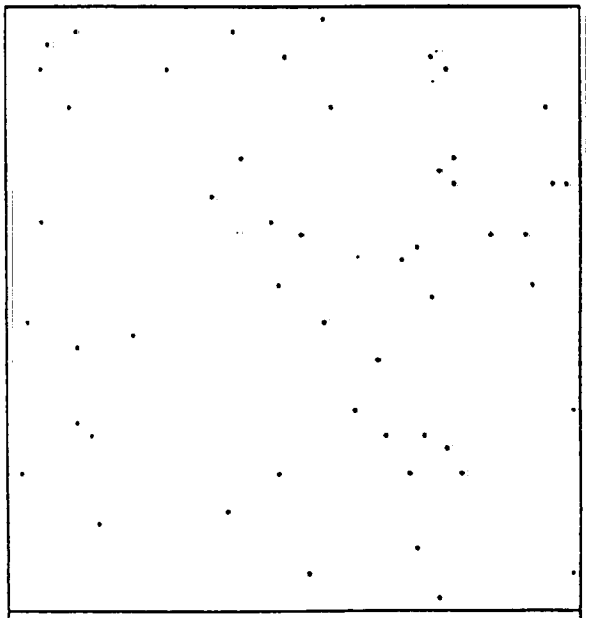

CHART #5
TYPICAL INDOOR AIR WITH VENTILATION INCREASED TO 20 CFM/PERSON AND NO SMOKING

## CONCLUSION

The Visual Indoor Air computer modeling program will produce dot charts which allow for easy comparison of indoor air quality parameters. It is set up so that if actual measurements of a specific situation are available these values can be entered into the program to model a real life situation. In the absence of these measurements, values obtained from literature sources can be readily used to model hypothetical situations.

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Source: https://www.industrydocuments.ucsf.edu/docs/zqyx0000

## REFERENCES


1. Oldaker, G.B., Perfetti, P.F., Conrad, F.C. Jr., Conner, J.M. and McBride, R.L. 1990. Suppl. Int. Arch. Occup. Env. Health. 99-104.
2. Wadden, R.A. and Scheff, P.A. 1983. Indoor Air Pollution, Characterization, Prediction and Control. John Wiley & Sons. New York, NY. 213 pp.
3. Badre, R., Guillerm, R., Abran, N., Bourdin, M. and Dumas, C. 1978. Annales Pharmaceutiques Francaises. 36(9-10), 443-452.
4. Bayer, C.W. and Black, M.S. 1987. Biomedical and Environmental Mass Spectrometry. 14(8), 363-367.
5. Eatough, D.J., Lewis, L., Lamb, J.D., Crawford, J., Lewis, E.A., Hansen, L.D., and Eatough, N.L. 1988. Proc. 1988 EPA/APCA Int. Symp. on Measurement of Toxic and Related Air Pollutants. pp. 104-112.
6. Lofroth, G., Burton, R.M., Forhand, L., Hammond, S.K., Sella, R.L., Zweidinger, R.B. and Lewtas, J. 1989. Environ. Sci. Technol., 23(5), 610-614.
7. National Academy of Science. 1986. Environmental Tobacco Smoke, Measuring Exposures and Assessing Health Effects. National Academy Press. Washington D.C. 337 pp.
8. Sterling, T.D., Collett, C.W., Sterling, E.M. 1987. J. Occup. Med., 29(1),57-62.
9. Sterling, T. 1988. Proc. Indoor Amb. Air Qual. Conf. London, pp. 89-98.
10. Sterling, T.D., and Mueller, B. 1988. Am. Ind. Hyg. Assoc. J., 49(9), 423-426.
11. U.S. Surgeon General. 1986. The Health Consequences of Involuntary Smoking. U.S. Department of Health and Human Services, Rockville, MD. 332 pp.
12. Bardana, E.J. Jr., Montaro, A. and O'Hollaren, M.J. 1988. Clin. Rev., 11., 6, 61-89.
13. Nero, A.J. Jr. 1988. Sci. Am., 258(5), 42-48.
14. Samet, J.M., Marbury, M.C. and Spengler, J.D. 1987. Am. Rev. Respir. Dis., 136, 1486-1508.
15. Samet, J.M., Marbury, M.C. and Spengler, J.D. 1988. Am. Rev.Respir. Dis., 137, 221-242.
16. Godish, T. 1989. Am. J. Public Health. 79, 1044-1045.
17. Yocum, J.E., 1982. JAPCA. 32(5), 500-520.
18. Good, B.W., Vilcins, G., Harvey, W.R., Clabo, D.A. and Lewis, A.L. 1982. Env. Int. 8, 167-175.
19. Arundel, A., Sterling, T. and Weinkam, J. 1988. Proc. Indoor Ambient Air Qual. Conf. London. pp. 242-251.
20. Tancrede, M., Wilson, R., Zeise, L. and Crouch E.A. 1987. Atmospheric Environment. 21(10), 2187-2205.
21. U.S. Environmental Protection Agency. 1987. Total Exposure Assessment Methodology (TEAM) Study: Summary and Analysis. Volume 1. Office of Research and Development. U.S. Environmental Protection Agency. Wash. D.C. 209 pp.
22. Muramatsu, M.E., Umemura, S., Fukui, J., Arai,T. and Kira, S. 1987. Int.Arch. Occup. Environ. Health. 59, 545:550.
23. Hinds, W.C. and First, M.W. 1975. New Eng. J. Med., 292(16), 844-845.
24. Kirk, P.W., Hunter, M., Baek, S.O., Lester, J.N. and Perry, R. 1988. Proc. Indoor Amb. Air Qual. Conf. London. pp. 99-112.
25. Mattson, M.E., Boyd, G., Byar, D., Brown, C., Callahan, J.F., Corle, D., Cullen, J.W., Greenblatt, J., Haley, N.J., Hammond, S.K., Lewtas, J., Reeves, W. 1989. JAMA. 261(6), 867-872.
26. Muramatsu, M., Umemura, S., Okada, T. and Tomita, H. 1984. Env. Res., 35, 218-227.
27. Oldaker G.B. and Conrad, F.C. 1987. Environ. Sci. Technol., 21, 994-999.
28. Occupational Safety and Health Administration. 1989. Air Contaminants - Permissible Exposure Limits. U.S. Department of Labor. Occupational Safety and Health Administration. 84 pp.
29. Baker, R.R., Case, P.D. and Warren, N.D. 1988. Proc. Indoor Ambient Air Qual. Conf. London. pp. 121-130.


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Source: https://www.industrydocuments.ucsf.edu/docs/zqyx0000

# ERRATUM

Pedelty, J.F. and Holcomb, L.C. 1990. Env. Technol. 11,1053-1062.

The equation:

$$Ci=\frac{K[(Q(1-F)(Co))+S)][1-2.7183^{-K(Q+Q1F)T}]+[Cs(2.7183^{-K(Q+Q1F)T}]}{K(Q+Q1F)}$$

on page 1055 should be changed to read:

$$Ci=\frac{[K(Q)(1-F)(Co)]+S[1-2.7183^{-K(Q+Q1F)T}]+[Cs(2.7183^{-K(Q+Q1F)T})]}{K(Q+Q1F).}$$

This is a typographical change in the text only. The results based on the model are not affected.

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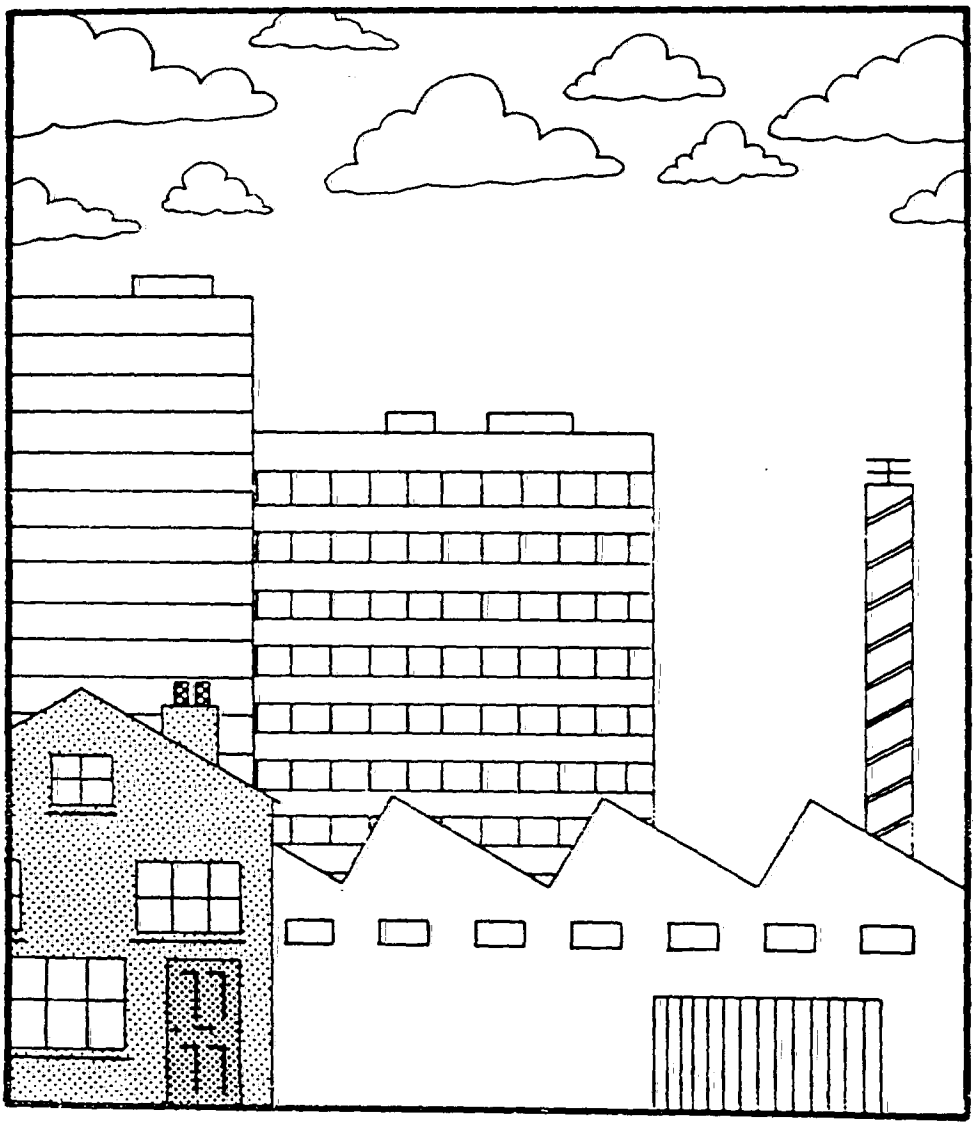
# INDOOR AND AMBIENT AIR QUALITY



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Edited by R. Perry
and P.W. Kirk

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# ASHRAE: ADDRESSING THE INDOOR AIR QUALITY CHALLENGE WITH ENERGY CONSCIOUS DESIGN

H.E.B.Burroughs

ASHRAE Inc., 1791 Tullie Circle NE, Atlanta, GA 30329, USA

ABSTRACT

The paper reviews how the issue of poor indoor air quality is affecting the heating, ventilation, air-conditioning, and refrigerating industry and emerging technology; the position of the American Society of Heating, Refrigerating and Air-Conditioning Engineers (ASHRAE) on indoor air quality; the impact of ASHRAE's revision of Standard 62, "Ventilation for Acceptable Indoor Air Quality," which is expected to be approved in 1988; the importance of the indoor air quality issue in the United States; recently approved and pending legislative and regulatory activities; and ASHRAE's role in providing safe and healthy environments in regards to indoor air quality.

It is a pleasure and honor to join you at this International Conference on Indoor & Ambient Air Quality. The interest of the American Society of Heating, Refrigerating and Air-Conditioning Engineers (ASHRAE) in indoor air quality goes back to the formation of the organization's predecessor society in 1895. Today, the activities of ASHRAE in the area of indoor air quality (IAQ) and ventilation include research, standards-writing, and education through seminars, conferences, speaking engagements, and publications.

In 1895, the recommended ventilation rate was 30 cubic feet per minute (cfm) of air per person. This amount of outdoor air was found to be necessary to control human body odor in buildings. Odor, a primary component of air quality was the issue. As personal hygiene improved in the early part of this century, ventilation rates were reduced.

Recently, IAQ has been a subject of increased concern because indoor contaminant levels and exposures frequently exceed those encountered outdoors, many new products are being introduced into the indoor environment that provide increased levels of exposure, and energy conservation measures that reduce ventilation rates can elevate indoor contaminant concentrations. Let me note that inadequate ventilation is not a source of indoor contaminants but, rather, allows contaminant levels from existing sources to be elevated.

Researchers have learned that indoor air contaminants may affect occupants' health by causing discomfort, annoyance, or even well-defined diseases. These illnesses result in reduced employee productivity and thousands of dollars in lost manhours. When the sources of indoor pollution are identified and the problems corrected, the pattern of increased employee absenteeism is immediately reversed. Initially, the problem is to identify the sources of indoor air contaminants. Contaminants may originate from the building's furnishings and materials, equipment operation, and occupants and their activities.

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An additional major source of indoor pollution may be the outdoor air which is taken into a building. The results of an indoor air quality study conducted by the National Institute for Occupational Safety and Health found that in 11 percent of the 446 buildings investigated the source of the poor air quality was the outdoor air. The intake air may be polluted with carbon monoxide, nitrogen dioxide, and hydrocarbons from automobile exhaust, as well as hydrocarbon exhaust from commercial kitchens.

From my experience of environmental engineering in heavy industry, some conclusion can be drawn from the accumulation of knowledge in this technology that address indoor air quality:

* Adequate Make-Up Air. Used for positive pressurization, large quantities of make-up air were necessary to insure against infiltration from structural leakage and door openings and traffic; large equals 20 to 40 percent and as much as 100 percent outside air. What does this tell us about the IAQ problem of inadequate ventilation air for indoor air quality?
* Treated Outside Air. Extensive filtration treats outside air to preclean it from gas phase and particulate contaminants; often, dropping sulphur concentrations by three orders of magnitude from part per million range to low part per billion range. Is your outside make-up air really clean enough to provide contaminant reduction through dilution or are you merely re-entraining more of different contaminants?
* High Internal Air Change Rates. Cleansing air to fractional part per billion contaminant range requires repeated and constant scrubbing by sorption and filtering units. Sometimes 10 and 20 air changes are not unusual. What does this say to us about VAV systems with "zero" air change or the whole issue of ventilation efficiency?
* Humidity Control. The conditioned space requires tight humidity control in the 50 to 55 percent range, but also demands very low variations of relative humidity, no more than two percent variation an hour. This gives us some clue as to the need to understand the inter-relationship of moisture content or air, either relative or absolute, with contaminant levels and perception phenomenon.

At the ASHRAE-sponsored conference IAQ 87, a case was cited in which outdoor air contaminated an office building, resulting in an employee absenteeism increase of 600 percent for two consecutive months compared to the same two-month period in the previous year. The employer was alarmed when manhour losses soared from 50 hours in April and May, 1985, to 300 to 350 hours in April and May, 1986. The outdoor air problem was caused by grease fumes emitted from a restaurant. After the restaurant's grease fumes were discharged, they were not diluted by being carried up above the building. This is fundamental in the prevention of indoor air quality problems. In downtown locations, exhaust fumes should be released at the highest point of the source building. After $25,000 in lost manhours in a five-month period, after a 600 percent increase in employee absenteeism in a two-month period, and after five months of negotiations, the restaurant owner agreed to install a new fan and ductwork. However, odors still persist to this day.

Building designers need to carefully evaluate the air flow over buildings and the air movement around buildings to minimize the amount of contaminated air entering occupied spaces. Once contaminated outdoor air has entered an office building, it will affect employee health, comfort, productivity, and absenteeism. The environmental controls for buildings need to serve three basic functional needs: comfort, health, and productivity.

Many cases of so-called "sick buildings" have been investigated, and physical causes for occupant complaints documented. Causes of poor indoor air quality include lack of system maintenance, changes to thermal and contaminant loads imposed during the lifetime of the building, changes in control strategies to meet new objectives during the lifetime of the building, and inadequate design of the original system. Problems of inadequate design include inadequate outdoor makeup air for ventilation, contaminated makeup air, poor air distribution within buildings and rooms, and unbalanced exhaust air.

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Once ventilation air has entered the building it must be properly distributed to occupants to avoid poor or bad ventilation. If portions of a building are leased to tenants, redesign of that space, such as erecting partitions or changing an executive office to a conference room, can dramatically affect the air distribution design or the required ventilation rate.

In 1973, ASHRAE published its first standard directly addressing ventilation: Standard 62-73, "Standard for Natural and Mechanical Ventilation." This standard specified ventilation rates using two sets of criteria: minimum ventilation rates to accommodate energy conservation (typically 5 cfm per person) and recommended ventilation rates for comfort in odor-free environments (typically 15 cfm per person). Other important considerations included the recognition of recirculation air and filtration systems for ventilation and the need for cleaning outdoor air when its quality was not acceptable. No mention was made, however, of the the nature of indoor pollutant sources and their impact on indoor air quality.

In response to concerns about energy consumption in buildings, in 1975 the organization published ASHRAE Standard 90-75, "Energy Conservation in New Building Design." Standard 90, which has been adopted in whole or in part by building codes in all 50 states in the United States, recommended that the minimum ventilation rate in Standard 62 be used for design purposes. This requirement had the effect of eliminating the established recommended values for ventilation and caused concern that the quality of indoor air would be compromised. When Standard 62 was revised and published in 1981 it listed required ventilation rates for nonsmoking and smoking areas rather than minimum and recommended values. Other criteria in addition to no smoking were required for designing a system utilizing 5 cfm, such as special air cleaners. A no smoking environment was, by itself, not sufficient for 5 cfm.

ASHRAE Standard 62-1981 is now being revised and is expected to be published this year. It is helpful to consider some of the major changes that have been made and the rationale for these changes. The distinction between smoking permitted and smoking prohibited has been abandoned, therefore reaffirming ASHRAE's recommended ventilation rates. The proposed revision to Standard 62 recommends the same cfm for office spaces as the existing standard recommends for that same space if any smoking is allowed. After all, how many office buildings are truly managed and operated as strictly non-smoking buildings?

Research undertaken since 1981 shows that occupants have a negative response to CO2 at a room concentration of 0.25 percent (the level with 5 cfm). Previously, it was assumed from experience with submarines, industrial settings, and bomb shelters that healthy people could tolerate 0.5 percent CO2. On that basis, it was thought that 0.25 percent CO2 was a sufficient safety margin.

More recent studies show, however, that the space will develop considerable odors and occupants will find the atmosphere stuffy and too warm. It is necessary to reduce the CO2 level to 0.10 percent or lower to avoid these effects. The World Health Organization, European countries, and Japan all have adopted 0.10 percent CO2 as the recommended limit. This requires 15 cfm per person of outdoor air. At this ventilation a minimum amount of tobacco smoke can be tolerated. If we assume that we are going to satisfy only 70 percent of the population who are nonsmokers, and that the smokers (assuming 30 percent of the occupants) are smoking at a rate of 1.7 cigarettes per hour (a minimum amount of smoking), the 15 cfm of outdoor air is required.

Can filters still be used to reduce the amount of ventilation air? The recommended 15 cfm is the amount of ventilation required to dilute contaminants to acceptable levels. Therefore, it is necessary that at least this amount of air be delivered to the conditioned space whenever the building is in use except for times of intermittent or variable occupancy. Properly cleaned air

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may be circulated. But, for other than intermittent or variable occupancy, the portion of outdoor air should not be less than 15 cfm per person.

The proposed revision to Standard 62 specifies rates which vary from a low of 15 cfm per person (for areas such as hotel dormitory sleeping areas, lobbies, and office reception areas) to 60 cfm per person for smoking lounges. A number of applications in the proposed revision of the standard were reduced from 25 cfm per person as specified in the 1981 document to 20 cfm.

ASHRAE Standard 62-1981, although admittedly not comprehensive, remains the most generally recognized standard in the United States for providing ventilation for acceptable indoor air quality. It is, therefore, appropriate to discuss how the standard should be used to achieve its purpose of specifying minimum ventilation rates and indoor air quality that will be acceptable to human occupants, which are intended to avoid adverse health effects. It should be noted that an ASHRAE standard has no mechanism for enforcement. Provisions of the standard must be written into a building code before the standard can be enforced. It represents the collective judgement of people active in the technology being addressed as to what is a realistic and acceptable level of performance.

ASHRAE supports and encourages the use of voluntary consensus standards by the public sector. A major objective of the Society is to establish a formal procedure for obtaining effective participation of public-sector organizations in the identification of the need for specific standards and in the development of those standards. Standard 90, used as a consensus resource document in building codes in all 50 states, is credited with reducing energy consumption in commercial buildings by 25 percent. One of the reasons Standard 90 has had such an impact at the national, state, and local levels is because of the important participation of public sector and private sector in its development. It therefore follows that ventilation standards can be used to ensure acceptable indoor air quality, and a voluntary consensus process can effectively establish ventilation standards.

Another recommendation of ASHRAE's, as stated in its Position Paper on Indoor Air Quality, approved by the Board of Directors on August 1,, 1987, is that the revised Standard 62, when approved, be widely disseminated and referenced by building codes.

Increasing research is another recommendation presented in the position paper, which is guiding ASHRAE's indoor air effort. Of particular importance is research on health effects and control through engineered systems of radon, asbestos fibers, unvented combustion appliances, formaldehyde, tobacco smoke, and volatile organic compounds. Every year, ASHRAE invests more than one million dollars in research. The principles behind conducting the research program are that the results benefit all people, it is not just for the present but for the future, and it is not directly aimed at product development but instead at the discovery of generic scientific and engineering principles. Since 1969, ASHRAE has sponsored 14 research projects specifically on ventilation requirements and indoor air pollutants. The Society has set goals to be achieved within the next couple years which will increase its expenditures in research from 1.25 million to 2 million dollars, and which will triple research expenditures in the areas of occupant health and safety, including indoor air quality.

Findings from one such ASHRAE research project, "Indoor Air Quality Evaluation of Three Office Buildings," were presented at the third annual ASHRAE-sponsored indoor air quality conference, April 11-13, 1988, in Atlanta, Georgia. This was the first documented study of an office complex specifically constructed to minimize indoor air pollution. In the study, this complex was compared with two other similar office complexes that were not specifically designed to reduce pollutant levels. Little documented data on pollutant concentration in offices are available, making the findings of this study extremely important. The ability to field-measure existing levels of

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pollutants and to determine their concentration levels in operating conditions will aid in the assessment of health effects of various indoor pollutants.

An area in which federal government involvement is greatly needed is in research. ASHRAE specifically states in its position paper that support by the federal government for research in all non-proprietary areas of indoor air quality, including health effects, measurement methods, modeling, and control technology, should be substantially and selectively increased. The industry desperately needs research so that building designers, owners, landlords, employers, and tenants alike can know what acceptable indoor air quality is--when and whether you have it and how to correct deficiencies if they occur.

Legislation, which is taking a research oriented thrust, will help provide this data. Specific needs include:

* Exposure and dosage data and basic physiology information to establish target levels for known harmful contaminants.
* Ventilation efficiency...in both dilution effectiveness as well as dynamics of air distribution; system design, such as VAV boxes; and even dynamics of air from diffuser to the occupant in a room. In this regard we must dispel the myth of 5 cfm per person minimum and re-establish 15 cfm as minimum.
* Development of diagnostic and remedial protocols and practices for monitoring, diagnosing, or remedying buildings demonstrating sick building syndrome.
* Studies of occupant populations to establish cost effectiveness values and payback rates on the cost of health related absenteeism and loss of productivity.

The key to solving current and future indoor air quality problems will be research. Each year, the task of maintaining acceptable indoor air quality becomes more difficult as new chemicals are introduced into our indoor environments and as we learn more about the harmful effects of airborne contaminants.

Greater awareness has resulted in attitude changes which will make all our jobs easier:

* No Smoking. The recognition by management that smoking is inherently harmful to the electronics and to human health has resulted in a commitment to manage that exposure to environmental tobacco smoke.
* Focus on Long-Term Costs Not Short-Term Costs. The short-term cost of air scrubbing can range from 3 to 30 dollars per cfm, but the long-term cost is far greater than even replacing the computers, for it means lost production. How does this compare to building owners looking at short-term cost per rentable square footage going into a mechanical room, versus the long-term cost of losing an entire tenant floor or even owning a non-occupable "sick building."
* Maintenance Mentality. A strong commitment to preventative maintenance is mandatory to keep scrubbing units and mechanical systems operating at peak performance.

ASHRAE will continue to provide guidance for design, installation, operation, and maintenance; develop and promulgate standards; and encourage interactions with other groups that have similar objectives. To identify and characterize factors of the environment that are affected by heating, ventilation, air-conditioning, and refrigeration systems, equipment, operation, and maintenance and that have adverse effects on occupant health and comfort is a challenge continuously addressed by ASHRAE.

It is ASHRAE's concern that we maintain good indoor air quality through energy conscious design. But successful energy conservation and good indoor air quality are highly dependent on building performance. Mechanical systems must be properly maintained in order to provide continuing comfort, air quality, and operating economy.

The challenge will continue for building designers, owners, and operators to balance the quality of the indoor environment with low operating costs through energy efficiency.

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ASHRAE will respond to the need for improved air quality with improved, innovative, and cost-effective solutions. But we must present a uniform platform for addressing this "challenge of the decade." We are challenged to ensure that each individual works in an environment with acceptable indoor air quality, and challenged to ensure that our work force operates in safe and healthy surroundings.

# # #

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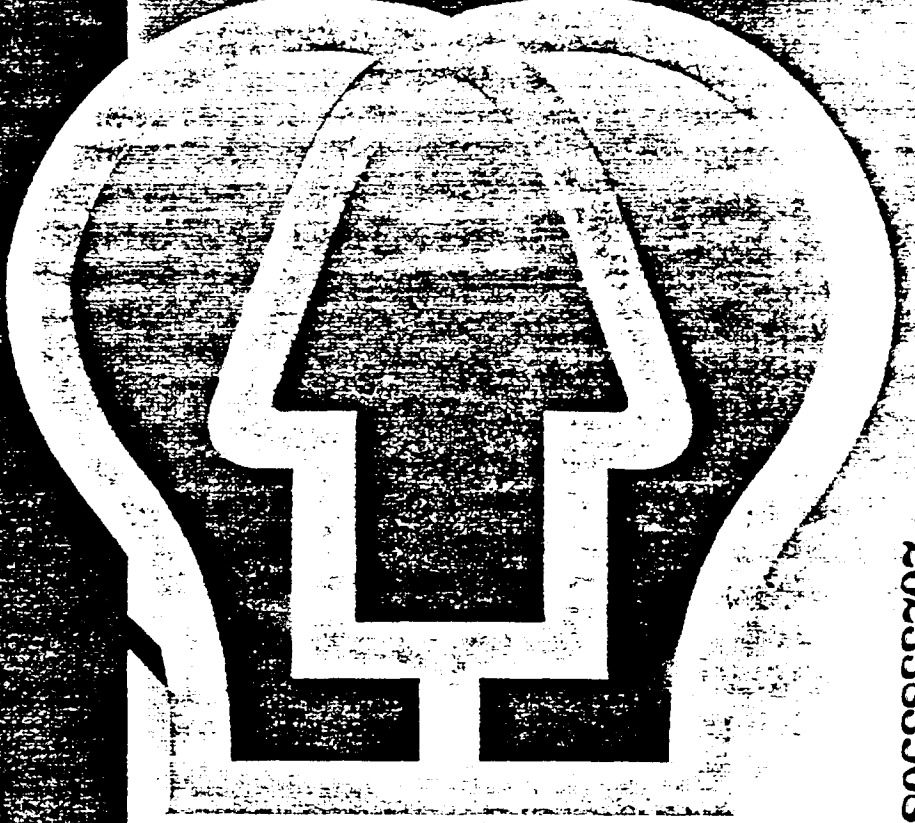
THE FIFTH INTERNATIONAL CONFERENCE ON INDOOR AIR QUALITY AND CLIMATE

TORONTO, CANADA
July 29 - August 3
1990



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# INDOOR AIR '90

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# THE HVAC COSTS OF INCREASED FRESH AIR VENTILATION RATES IN OFFICE BUILDINGS, PART 2

Joseph H. Eto
Lawrence Berkeley Laboratory
University of California
1 Cyclotron Road
Berkeley, California
USA 94720

This study reports on predicted changes in annual energy operating costs that result from increased minimum outside air ventilation rates. The analysis is based on parametric DOE-2.1C simulations for typical small and medium office buildings in ten U.S. cities. In the simulations, minimum ventilation rates are increased from 5.0 liters per second per person (L/s.person)(10 cfm/person) to 17.5 L/s.person (35 cfm/person). Annual building energy costs are calculated using current electricity and natural gas tariffs for each location. The results suggest that, for the buildings, climates, and economic conditions examined, increasing minimum outside air ventilation rates, from the lowest level to that called for by the current ASHRAE Standard (10 L/s.person) will have small impacts on annual building energy costs. We found an average of 5% for the small office and 3% for the medium office. These results are due to the relatively small amount of energy used for HVAC purposes in typical office buildings and the operation of an economizer cycle, which has the effect of increasing outside air ventilation beyond the minimum for most operating hours.

## INTRODUCTION

In 1989, ASHRAE adopted revisions to its Standard 62-1981, "Ventilation for Acceptable Air Quality" (1). The Standard offers two methods for compliance: a prescriptive method, which provides guidelines for designing a building for acceptable indoor air quality (through specification of minimum outside air ventilation rates), and a performance method, which relies on measurements of the completed building to determine indoor air quality. In this paper, we focus on changes in annual energy use and annual energy operating costs that result from simulations of typical office buildings operated to follow the guidelines of the prescriptive method.

In 1988, Eto and Meyer presented a similar study using a building energy simulation program to estimate the impacts of different ventilation rates for a large office building (2). They found that increasing minimum outside air ventilation rates from the lowest level of the earlier Standard (2.5 L/s.person, assuming no smokers) to the current Standard (10 L/s.person) would increase energy operating costs by no more than 5% and building first costs by no more than 1%. The current study is intended to complement this earlier analysis, by extending the range of building types and minimum outside air ventilation rates examined.

## METHOD OF ANALYSIS

The method of analysis relies on a series of parametric building energy simulations in which all features of the building are held fixed, except the minimum outside air ventilation rate. The

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other aspects of the building description, including structural, architectural, mechanical, and electrical characteristics and hours of operation and temperature setpoints, remain unchanged, not only as the minimum ventilation rate changes for a given city, but also across cities. This latter step ensures that results can be compared, on a consistent basis, between cities as well as within them.

Six simulations are performed, each with a different rate of minimum outside air ventilation. The lowest ventilation rate was 5.0 L/s.person (10 cfm/person) increasing in increments of 2.5-L/s.person (5 cfm/person) to 17.5 L/s.person (35 cfm/person). In normal operation, these minimum ventilation rates are frequently exceeded when, for cooling purposes, additional outside air is taken in through an economizer cycle.

The DOE-2 building energy analysis program (version DOE-2.1C) is used to study the changes in energy use, energy costs, and equipment sizing that result from increasing minimum outside air ventilation rates. The DOE-2 program was developed for the Department of Energy to provide architects and engineers with a state-of-the-art tool for estimating building energy performance (3). The DOE-2 program has been extensively validated (4).

The two office building prototypes simulated are based on actual buildings of recent vintage, with modifications that make them representative of typical 1980s building construction practice. The prototypes were originally developed for the ASHRAE-sponsored evaluation of revisions to Standard 90 (5). In that evaluation, the building was slightly altered for each climate; for the present analysis, only one building was used (designed originally for the Washington, DC, climate) for each location. Operating schedules were taken from the Standard Building Operating Conditions developed for the Building Energy Performance Standards (6). The HVAC system for the medium office building was designed so that only electricity would be used for cooling and only natural gas would be used for heating (of course, electricity is also used for lighting, fans, pumps, etc.). For the small office building, electricity provides both heating and cooling. Major features of the office building prototypes are summarized in Table 1.

The simulations are performed using weather data and current utility tariffs from ten U.S. cities. The weather data are from either the Weather Year for Energy Calculation (WYEC) series developed for ASHRAE (7), or from the Typical Meteorological Year series developed by NOAA (8).

## RESULTS

Table 2 summarizes the percentage changes in annual energy operating costs found for each minimum outside air ventilation rate and building type. The Table also presents the nominal annual operating cost (in $1987 per square meter) for each location and building type, at the base case ventilation rate of 5 L/s.person (10 cfm/person).

We find that increasing minimum outside air ventilation rates from the base case (5 L/s.person, 10 cfm/person) to the level called for by the new Standard (10 L/s.person, 20 cfm/person) increased energy operating costs by an average of 5% for the small office and 3% for the medium office. The greatest percentage increase in annual energy cost is found for the small office in Boston (9%) and Minneapolis (8%). For the medium office, the largest percentage increase is found in Miami (6%) followed by Washington (5%).

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The findings for the small office are driven by the large heating requirements found in Minneapolis and Boston (5 MJ/m\s-2\u2\d\s+2 and 4 MJ/m\s-2\u2\d\s+2 at 5 L/s.person, respectively). The findings for the medium office in Miami are driven largely by increases in cooling energy of 13% from a base case of 0.8 kWh/m\s-2\u2\d\s+2. For the medium office in Washington, a moderate increase in heating energy use (up 5% from a base case of 2 MJ/m\s-2\u2\d\s+2), coupled with relatively large increase in cooling energy (up 12% from a base case of 0.4 kWh/m\s-2\u2\d\s+2) explains the increase in total costs.

## DISCUSSION

The results presented in the previous section indicate that, for the simulations performed, increased minimum outside air ventilation rates will have relatively small effects on annual building energy costs. In particular, the simulations do not support a one-to-one relationship between percentage increases in minimum outside air ventilation and increases in annual energy costs. For example, a doubling in the minimum ventilation rate from the lowest value examined to that called for by the current Standard, corresponds to an average increase in annual energy costs of about 5% for the small office.

The primary reason for small increases in energy costs is that energy use for heating, cooling, and auxiliary HVAC end uses represents only a fraction of the total energy operating costs of modern office buildings. Energy use for lighting and miscellaneous equipment constitutes a large, fixed component of energy costs that is unchanged by increased outside air ventilation rates.

A second reason for our findings has to due with the operation of modern HVAC systems. In all of our simulations, we assume that an economizer is able to introduce outside air in excess of the minimum ventilation rate whenever the outside air temperature is less than a given value (specifically, we have used a drybulb setpoint of 19°C or 66°F. For most office buildings, normal operation of the economizer means that outside air ventilation rates will generally exceed the minimum rates called for in the Standard. That is, increased minimum ventilation rates can only increase energy use when the supply air temperature would otherwise be higher (in the heating mode) or lower (in the cooling mode), but for this minimum rate. In most office buildings, this circumstance only occurs at the extremes of the temperature scale, i.e., only at very low or very high outside air temperatures. Consequently, for a large number of operating hours, the Standard has no effect on energy use.

## SUMMARY

We have performed a simulation-based analysis of the increases in energy use and energy costs that result from building operation at different minimum outside air ventilation rates. The analysis relied on parametrically increasing minimum outside air ventilation rates for a medium and small prototypical office building in 10 U.S. cities. A minimum outside air ventilation rate from the previous Standard, 5.0 L/s.person (10 cfm/person), was the basis for comparison to both the current Standard of 10 L/s.person (20 cfm/person) and several higher minimum rates. Economics were evaluated with actual and current utility rate tariffs.

The results suggest, for the prototypes, climates, and economic conditions examined, that the increased minimum outside air ventilation rates called for by the new Standard: 1. May

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increase annual energy operating costs on average between 3% and 5% (for medium and small office buildings, respectively); 2. May increase energy costs relatively more for smaller buildings located in colder climates; and, 3. May increase energy costs relatively more for larger buildings located in warmer climates.


## ACKNOWLEDGMENT

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## REFERENCES


1. ASHRAE. 1989. "ASHRAE Standard 62-81: Ventilation for acceptable indoor air quality." Atlanta: American Society of Heating, Refrigerating, and Air-Conditioning Engineers, Inc.

2. Eto, J., and Meyer, C. 1988. "The HVAC costs of increased fresh air ventilation rates in office buildings." ASHRAE Transactions, V. 94, Pt.2.

3. Curtis, R., Birdsall, B., Buhl, W., Erdem, E., Eto, J., Hirsch, J., Olson, K. and Winkelmann, F. 1984. "DOE-2 building energy use analysis program." Lawrence Berkeley Laboratory, LBL-18046.

4. Diamond, S., Cappiello, C. and Hunn, B. 1986. "DOE-2 verification project, final report." Los Alamos National Laboratory, 10649-MS.

5. Battelle Pacific Northwest Laboratories. 1983. "Recommendations for energy conservation standards and guidelines for new commercial buildings, Volume III, Appendix A: Testing assumptions and inputs." U. S. Department of Energy, DOE/NBB-0051/7.

6. U. S. Department of Energy 1979. "Standard building operating conditions." DOE/CS-0118.

7. Crow, L. 1981. "Development of hourly data for weather year for energy calculations (WYEC), including solar data, at 21 stations throughout the U.S." ASHRAE Transactions.

8. National Climatic Center. 1981. "Typical meteorological year, user's manual." National Climatic Center.


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Source: https://www.industrydocuments.ucsf.edu/docs/zqyx0000

**Table 1. Summary of Building Characteristics**

| | Medium Office | Small Office |
|---|---|---|
| Size | 4,524 $m^2$ (48,680 $ft^2$) | 209 $m^2$ (2,250 $ft^2$) |
| Shape | 3 floors, rectangular | 1 floor, square |
| Construction | steels frame superstructure, 4" precast concrete walls | wood frame, brick veneer |
| Glazing | 36% of wall area, equally distributed | 50% north and south, 10% west, 3% east |
| Operation | 8 am - 6 pm weekdays, with some evening work, 30% occupancy on Saturday, closed Sundays and holidays | identical to medium office |
| Thermostat Settings | 24°C (76°F)cooling<br>22°C (72°F) heating (night and weekend setback)<br>17°C (62°F) | identical to medium office |
| Internal Loads | 26 $W/m^2$ (2.4 $W/ft^2$) lighting<br>5.4 $W/m^2$ (0.5 $W/ft^2$) equipment | identical to medium office |
| Occupancy | 13.7 $m^2$/person (148 $ft^2$/person) | 11.0 $m^2$/person (118 $ft^2$/person) |
| HVAC Air-Side | dual-duct system with variable speed fan; dry bulb economizer set at 19°C (66°F) | variable air volume, direct expansion, rooftop unit; dry bulb economizer set at 19°C (66°F) |
| Heating Plant | gas-fired hot water boiler (eff.=75%) | baseboard electric |
| Cooling Plant | air-cooled, hermetic reciprocating chiller (COP = 2.4) | direct expansion (COP=2.8) |



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**Table 2. Annual Energy Operating Cost Impacts**

| | 5 L/s.person (1987$/m$^2$) | Percentage Increase from 5 L/s.person | | | | |
|---|---|---|---|---|---|---|
| | | 7.5 L/s.person | 10 L/s.person | 12.5 L/s.person | 15 L/s.person | 17.5 L/s.person |
| *Small Office* | | | | | | |
| Atlanta | 0.16 | 2.0 | 4.0 | 6.4 | 8.7 | 11.3 |
| Boston | 0.35 | 3.8 | 8.5 | 14.0 | 19.0 | 25.3 |
| Chicago | 0.41 | 1.3 | 2.8 | 5.4 | 8.0 | 10.9 |
| Dallas | 0.13 | 1.9 | 3.7 | 5.8 | 8.3 | 11.3 |
| Miami | 0.21 | 1.9 | 3.5 | 5.4 | 7.1 | 9.0 |
| Minneapolis | 0.14 | 3.6 | 7.4 | 11.9 | 16.0 | 20.6 |
| New York | 0.27 | 2.7 | 5.8 | 9.5 | 13.1 | 17.4 |
| San Diego | 0.17 | 1.3 | 2.5 | 3.8 | 5.0 | 6.5 |
| Seattle | 0.06 | 2.9 | 6.2 | 10.2 | 14.3 | 19.1 |
| Washington | 0.25 | 1.2 | 2.3 | 3.7 | 5.0 | 6.5 |
| *Medium Office* | | | | | | |
| Atlanta | 0.13 | 2.1 | 4.1 | 6.2 | 8.2 | 10.4 |
| Boston | 0.13 | 1.6 | 3.2 | 4.9 | 6.8 | 9.2 |
| Chicago | 0.13 | 1.6 | 3.1 | 4.8 | 6.8 | 9.1 |
| Dallas | 0.14 | 2.1 | 4.2 | 6.4 | 8.6 | 10.8 |
| Miami | 0.14 | 2.8 | 5.7 | 8.7 | 11.8 | 14.9 |
| Minneapolis | 0.14 | 1.2 | 2.9 | 5.0 | 7.5 | 10.2 |
| New York | 0.13 | 1.8 | 3.5 | 5.2 | 7.3 | 9.6 |
| San Diego | 0.12 | 0.9 | 1.7 | 2.5 | 3.3 | 4.2 |
| Seattle | 0.12 | 0.6 | 1.1 | 2.0 | 2.8 | 3.8 |
| Washington | 0.13 | 2.3 | 4.6 | 7.0 | 9.5 | 12.0 |



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# Impact of "Designated Smoking Area" Policy on Nicotine Vapor and Particle Concentrations in a Modern Office Building

William M. Vaughan
Environmental Solutions, Inc.
St. Louis, Missouri

S. Katharine Hammond
University of Massachusetts Medical School
Worcester, Massachusetts

A series of measurements of nicotine vapor and particle concentrations were carried out in a modern office building. The measurement program was designed to document the impact of the new smoking policy (e.g. smoking restricted to two specific areas) on air quality in various parts of the building.

Measurements at over 30 locations examined areas where smoking was restricted, where smoking was permitted, and where an air handler shared the load of a designated smoking area. These measurements were taken at the same locations before and after the policy was implemented.

The measurement results indicated that:

- After the policy was implemented, general employee exposure to nicotine vapor (as a surrogate for all environmental tobacco smoke) was reduced by about 98 percent on most floors. Other patterns related to spillover from designated smoking areas and changes in the designated smoking areas themselves are described in the paper.
- Substantial reductions in exposure to nicotine vapor were found even for those general office areas which had the highest concentrations after the policy was implemented.

A large corporation implemented a new smoking policy in its headquarters in January 1988. This new policy restricted smoking to designated areas of the 32nd floor snack bar and the eastern part of the cafeteria only.

### Implications

The study reported in this article demonstrates that:

- It is possible to make objective measurements of nicotine vapor in an office setting using inexpensive passive sampling devices.
- It is possible to demonstrate the effectiveness of a change in smoking policy using before and after monitoring.
- Use of a designated smoking area can significantly (90–98 percent) reduce office exposure to nicotine vapor and hence other smoking vapors.
- With common air handlers typical of modern office buildings, adjacent office spaces can receive spillover of vapors from a designated smoking area.

In order to document the air quality impact of this change in policy, measurements were conducted on four floors of the headquarters before and after the policy went into effect. Nicotine vapor measurements documented the specific changes in air quality due to tobacco smoke. Additional measurements of inhaled particle load at selected locations linked particle and nicotine vapor concentrations (to assess changes in visible smoke as well).

The general objectives were to:

- Define the initial concentrations of nicotine vapor and inhalable particles (as a measure of baseline air quality).
- Document the change in air quality on a typical floor where smoking would no longer be allowed.
- Document the change in air quality in the 32nd floor snack bar (where smoking was allowed) as well as any "spill over" to adjacent office areas.
- Document the change in air quality on the 31st floor, which shares the air handler with the 32nd floor, to see if smoking impact was "imported" to this non-smoking area.
- Document the changes in air quality of the different areas of the cafeteria.

## Methods

### Sampling Devices

Two types of sampling devices were used in this study to capture the nicotine vapors from the air—passive and active. They both operated with the same principle of stabilizing the nicotine on a filter treated with sodium bisulfate. The passive devices utilized diffusion across a wind screen membrane to deposit the vapors. The active devices used a pump to draw room air through an identically treated filter at the rate of about 1.7 liters per minute. This active sampler was also equipped with a standard teflon coated glass fiber filter to test for inhalable particles at the same time, as explained below.

*Passive samplers.* The passive samplers consist of a plastic holder, a clip to hold it in place, a teflon-coated glass fiber filter treated with a 4 percent sodium bisulfate solution, and a plastic wind screen (to prevent strong drafts from influencing the sampling). Each filter holder is sealed until sampling begins when the solid lid is replaced by the wind screen. A schematic drawing of a passive sampler assembly appears in Figure 1.

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Each passive sampler was exposed from Monday morning through Friday afternoon to collect a weekly average nicotine concentration. It samples at a rate of about 24 mL/min as determined by chamber studies.[1] The sampling volume is then computed for the exposure interval by multiplying by the rate (24 mL/min) by the number of minutes of exposure.

*Active samplers.* Active sampling occurred on the Wednesday of each sampling period. The active sampler assembly is a typical occupational health sampling unit equipped with a standard particle filter ahead of a second treated filter to trap the nicotine vapor.[2] The inlet to the filter assembly was a 10-mm nylon cyclone that is designed to allow only the respirable size particles to pass to the filter at a sample rate of 1.7 liters/minute (L/m).

The sample volume for both filter elements is determined by the sample rate which was maintained by battery operated SKC personal sampling pumps (Model 224). These pumps were calibrated at 1.0 and 2.0 L/min before each sampling period. The flow meter reading on each pump was used to adjust the sampling rate to about 1.7 L/m. These flows were checked at two- to three-hour intervals during the intensive sampling days to maintain constant flow. Little adjustment was ever required.

### Sampling Locations and Conditions

Phase I of the sampling was conducted in early December 1987, four weeks prior to the implementation of the policy restricting smoking. Phase II sampling occurred in February 1988, after the policy had been in effect for seven weeks.

### Site Selection Procedure

The sampling locations were chosen to determine changes in both typical work area exposure and in areas to be most heavily impacted by the new policy. The 32nd floor was selected since smoking would be allowed in the snack bar resulting in an influx of smokers from the rest of the building. The 31st floor was selected since it shares an air handler with the 32nd floor and might share any decreased air quality associated with an increase in smoking on the 32nd floor. The 19th floor was selected as a "typical" floor which would experience only a decrease in its smoking population. The cafeteria was selected since it would be divided into smoking and non-smoking areas.

Each of the office floors is primarily open with cubicles defining work space. Only one "private" office was sampled (location # 22 below). The specific sample locations on these office floors were determined by generating random number grid coordinates for about 2,000 sq-ft sections of each floor. This step resulted in different random locations for possible sampling on each floor. This pattern was then compared to the information on office cubicles occupied by smokers (S) and non-smokers (NS) or vacant (V), as supplied by floor managers. Possible sample locations were then shifted one or two cubicles away to assure a mix of S, NS and V samples from each floor. The snack bar on the 32nd floor received three samplers, since it would experience the largest potential change and would warrant the greatest attention.

Table I. Sample locations and types of exposure (sites with "a" designate active sampling also).

| Floor | Location | Type of Exposure |
|---|---|---|
| 32nd | 1 | Public-snack bar, smoking (Phase II) |
| | 2 | Public-snack bar, smoking (Phase II) |
| | 3[a] | Public-snack bar, smoking (Phase II) |
| | 4 | Non-smoker |
| | 5 | Non-smoker |
| | 6[a] | Vacant (Phase I), non-smoker (Phase II) |
| | 7[a] | Smoker (Phase I), vacant (Phase II) |
| | 8 | Smoker |
| | 9 | Non-smoker |
| 31st | 10[a] | Smoker |
| | 11 | Common terminal area |
| | 12 | Non-smoker |
| | 13 | Vacant (Phase I), non-smoker (Phase II) |
| | 14 | Public-on a divider |
| | 15[a] | Non-smoker |
| | 16 | Damaged (Phase I), non-smoker (Phase II) |
| | 17 | Vacant |
| 19th | 18 | Non-smoker |
| | 19 | Non-smoker |
| | 20 | Non-smoker |
| | 21 | Damaged (Phase I), non-smoker (Phase II) |
| | 22[a] | Smoker (enclosed office) |
| | 23 | Smoker |
| | 24[a] | Non-smoker |
| | 26 | Damaged (Phase I), non-smoker (Phase II) |
| Cafeteria | 26[a] | Public, no smoking (Phase II) |
| | 27 | Public, no smoking (Phase II) |
| | 28[a] | Public, no smoking (Phase II) |
| | 29 | Public, no smoking (Phase II) |
| | 30 | Public, no smoking (Phase II) |
| | 31 | Public, no smoking (Phase II) |
| | 32[a] | Public, only Phase II—smoking area |

At the time the six locations for the Phase I cafeteria sampling were selected, there was no firm idea as to which areas would be designated smoking and which non-smoking. Hence, a relatively even distribution of locations (# 26 through # 31) was laid out in the western portion of the cafeteria. Ultimately, the eastern half of the cafeteria was designated the smoking area. For the Phase II sampling, an additional sample location (# 32) in that area was set up for the active sampling.

The final 32 sampling locations were refined during the setup for Phase I sampling. Usually a passive sampler was clipped to the center of a desk lamp or at the end of a lamp to avoid being directly over an ash tray. For the open areas, the passive samplers were attached to the drop ceiling supports in the snack bar and the light fixtures of the cafeteria.

During each phase of sampling, active samples were gathered at nine to ten locations. These active sites were also chosen arbitrarily to achieve a mix of S, NS and V sites throughout the three office floors. Two sites in the western half of the cafeteria were also selected.

Table I lists the final mix of sampling locations and indicates the type of exposure. Figures 2 and 3 show the locations of these sample points on floor plans for the two floors experiencing the greatest changes.

For each sampling location, a log sheet was completed which noted the actual installation point, indications of smoking in the area, time of placement and removal, and comments. Each site was revisited at least once during the Wednesday intensive sampling and on removal. These site-specific records assisted in interpreting the nature of the exposure or unique conditions at each location.

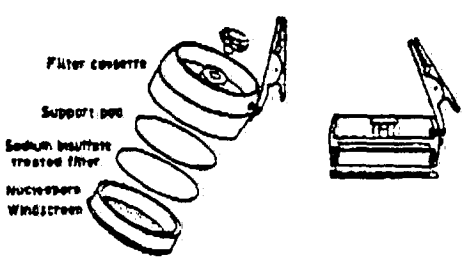


Figure 1. Diagram of passive monitor to sample nicotine in the air.

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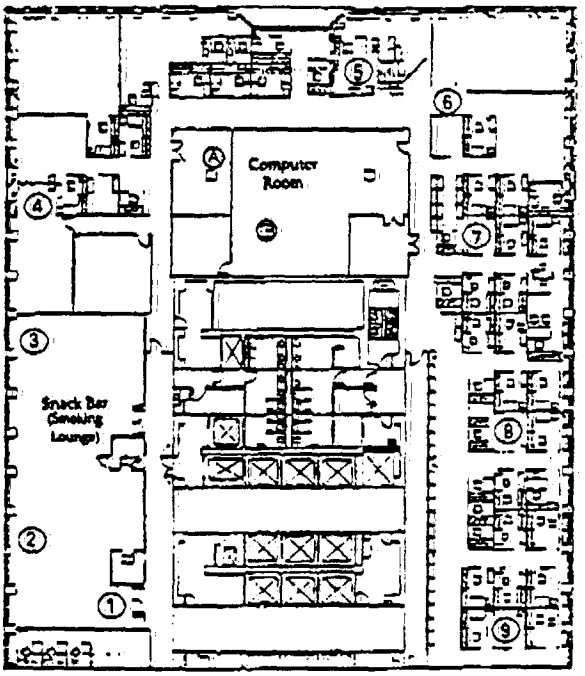


Figure 2. Map of sampling locations on 32nd floor.

### Sample Analysis

The nicotine vapor collected on both the passive and active filters was analyzed by the procedures described by Hammond and Leaderer.[1,2] Briefly, the nicotine was desorbed from the filters with water. The pH was adjusted with sodium hydroxide. The nicotine was concentrated by liquid/liquid extraction into ammoniated heptane. A small amount of the heptane solution was injected into a gas chromatograph which was equipped with a nitrogen selective detector.

The detection limit for the method depends on the nicotine levels of field blanks (filters shipped to the study area, opened briefly on site, and returned to the laboratory just like the actual samples) which must be subtracted from the levels obtained on actual samples. For this study the passive blanks were at or below 0.01 $\mu$g. The final passive detection limit was determined to be 0.03 $\mu$g of nicotine. For the active samples the detection limit was determined to be 0.2 $\mu$g of nicotine (partly because of a different range for analysis).

The particle loading is determined from the front filter of the active filter assembly. It is pre-weighed at the laboratory before being sent to the field, then re-weighed after sampling. The difference is then reported as the particle loading. The reliable minimum detection limit is about 20 $\mu$g of particle and all values below that limit are reported as not detected (ND) with the actual value shown in parentheses.

## Results and Observations

### Results

Tables II and III present a summary of all nicotine vapor analyses during the study. The office results are grouped by floor with a separate sub-grouping of public area (snack bar and cafeteria) samples for both before and after conditions. Reference should be made to Table I and Figures 2 and 3 for actual cubicles and general locations on each floor.

It should be noted that these passive sampling results are computed from a 45-hour exposure period assuming that, on the average, the sources of nicotine vapor and the people exposed are in the area about 9 hours a day, even though the passive sampling proceeds for 24 hours a day. This assumption may be inaccurate for some locations but is a best estimate that also allows comparisons with other work week studies by using a consistant data reduction approach.[3] In one instance, at site #10, the smoker was away from the office for three days during Phase I. We have adjusted the nicotine level at that location to reflect 18 hours of "exposure," not 45.

The nicotine values from the active samplers for each sampling phase are presented in Table IV. Columns allow comparisons of the intensive sampling on Wednesday with the "work week" average. This grouping shows whether the Wednesday samples were "typical" (i.e. Wednesday readings close to average week values) as indicated by nicotine levels, and most are typical.

Table V allows comparison of the before and after active samples for both nicotine and particle values.

### Observations

*Before conditions.* Nicotine vapor concentrations at nonsmoking desks in the general office areas were about 2 $\mu g/m^3$ in December 1987. Nicotine concentrations in the snack bar were about 11 $\mu g/m^3$ or just over 5 times NS desks, while those in the cafeteria were about 3.4 $\mu g/m^3$ or 1.5–2 times NS desks.

The range of average smoker desk concentrations on each floor were highly variable (Table III), and ranged from 3 to 33 $\mu g/m^3$ or about 2 to 15 times that at the NS desks.

In general, the "vacant" desk areas had about the same nicotine vapor concentration in December 1987 as the nonsmoker areas with the exception that the vacant space at Site #17 had a value of 6.7 $\mu g/m^3$. Perhaps there was a nearby smoker or a smoker borrowed the cubicle to work for several hours that week.

Unfortunately four of the Phase I particulate filters were lost so that adequate baseline information is missing. Those data that are available show low particulate levels at the NS and V desks, as would be expected, and higher values at one

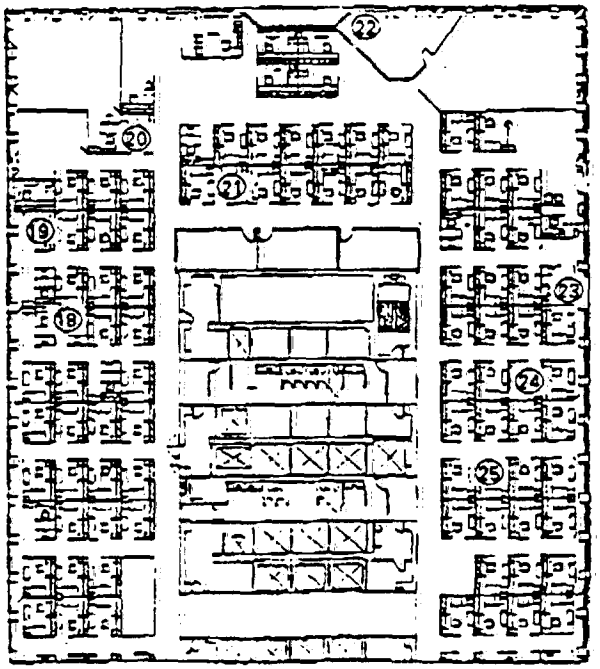

Figure 3. Map of sampling locations on 19th floor.

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Table IIa. Nicotine concentrations ($\mu g/m^3$) in offices (+ to left indicates Wed. intensive sampling).

Offices

32nd Floor

| Loc. # | Type SNV | Before | After | Change |
|---|---|---|---|---|
| 4 | N | 1.8 | 0.7 | −1.1 |
| 5 | N | 2.2 | 0.3 | −1.9 |
| +6 | V | 1.6 | 0.1 | −1.5 |
| +7 | S | 33.3 | 0.4 | −32.9 |
| 8 | S | 14.7 | 0.4 | −14.3 |
| 9 | N | 1.9 | 0.1 | −1.8 |
| | Area Ave. = | 9.3 | 0.3 | −9.0 |

31st Floor

| Loc. # | Type SNV | Before | After | Change |
|---|---|---|---|---|
| +10 | S | 3.4 | 0.7 | −2.7 |
| 11 | V | 3.1 | 0.4 | −2.7 |
| 12 | N | 1.8 | 0.4 | −1.4 |
| 13 | N | 2.1 | 0.3 | −1.8 |
| 14 | S | 4.6 | 0.3 | −4.3 |
| +15 | N | 1.9 | 0.3 | −1.6 |
| 17 | V | 6.7 | 0.2 | −6.5 |
| 16 | N | damaged | 0.2 | |
| | Area Ave. = | 3.4 | 0.4 | −3.0 |

19th Floor

| Loc. # | Type SNV | Before | After | Change |
|---|---|---|---|---|
| +22 | S | 4.3 | 0.2 | −4.1 |
| 18 | N | 0.9 | 0.0 | −0.9 |
| 19 | N | 1.6 | 0.1 | −1.5 |
| 20 | N | 5.3 | 0.1 | −5.2 |
| 23 | S | 3.9 | 0.0 | −3.9 |
| +24 | N | 1.0 | 0.1 | −0.9 |
| 21 | N | damaged | 0.0 | |
| 25 | N | damaged | 0.0 | |
| | Area Ave. = | 2.8 | 0.1 | −2.7 |

Table IIb. Nicotine concentrations ($\mu g/m^3$) in snack bar & cafeteria (+ to left indicates Wed. intensive sampling).

Public areas

Snack Bar

| Loc. # | Before | After | Change |
|---|---|---|---|
| 1 | 10.4 | 36.6 | 26.2 |
| 2 | 13.2 | 41.0 | 27.8 |
| +3 | 10.2 | 178.7 | 168.5 |
| Area Ave. = | 11.3 | 85.4 | +74.1 |

Cafeteria

| Loc. # | Before | After | Change |
|---|---|---|---|
| +26 | 5.0 | 2.4 | −2.6 |
| 27 | 5.8 | 3.5 | −2.3 |
| +28 | 6.7 | 4.1 | −2.6 |
| 29 | 5.5 | 3.3 | −2.2 |
| 30 | 2.4 | 6.6 | 4.2 |
| 31 | 1.8 | 11.6 | 9.8 |
| Area Ave. = | 4.5 | 5.3 | +0.8 |

smoker's desk. The western part of the cafeteria had quite low particulate levels during Phase I measurements.

*After conditions and comparisons.* Overall air quality, as measured by nicotine concentrations improved dramatically in the office areas as a result of the new policy restricting smoking. The average non-smoker desk values were 0.1–0.3 $\mu g/m^3$ during the February 1988 measurements compared to 2.0 $\mu g/m^3$ earlier. The greatest improvement in overall air quality (95+ percent reduction in ETS exposure) was on the 19th floor, due to the prohibition of all smoking without any common air handling with a smoking location.

However, there is evidence of some tobacco smoke "spillover" into non-smoking areas on the 31st and 32nd floors, which had reductions in ETS exposure of only 84 percent and 80 percent respectively. The highest levels at non-smoker desks were on the 32nd floor. The highest level at a 32nd floor cubicle was at Site #4 (0.7 $\mu g/m^3$) which receives fumes from the hallway outside the snack bar smoking area.

(There is also some evidence of "off gassing" of tobacco smoke vapors from the clothes of smokers or from the porous fabric on the divider walls. This observation comes from the general pattern that most smoker cubicles exhibit nicotine values which are slightly higher than non-smoker values.)

The 32nd floor snack bar experienced the most significant decrease in air quality in the building. The February 1988 average nicotine vapor concentration (85.4 $\mu g/m^3$) was 7.6 times higher than the already high December 1987 levels. These concentrations were highest at the eastern end of the "designated smoking area." (Site #3), perhaps due to ventilation changes made by the maintenance staff to encourage the air flow in that direction.

The highest nicotine vapor and particulate readings were at Site #3 in the snack bar. These levels were at least 1,800 times higher than the average values in the non-smoking office areas.

The cafeteria—in spite of its higher ceilings and larger air volume—showed, at best, only slight improvement in air quality at four NS locations (#26–#29) and decreased air quality at two locations (#30 and #31). The latter non-smoking locations were nearest the newly designated smoking area and were three to ten times higher in February 1988 than in December 1987. Due to the dramatic change in these two cafeteria nicotine levels, the overall western cafeteria (NS area) readings are 16 percent higher in February 1988 compared to December 1987. The second highest nicotine vapor (and particle readings) measured were in the newly designated smoking area at the eastern end of the cafeteria. Four of the six western cafeteria locations did show improvement in nicotine vapor levels in February 1988 compared to December 1987 but the reduction was only about 50 percent compared to 80–98 percent on the working floors.

[Note: Since this original work was performed in 1987 and early 1988, two subsequent studies have been performed in the same building on the "snack bar" floor (32nd) using the same sampling and analytical methods. One study in December 1988 focused on levels in and near the computer room on the 32nd floor that had a separate air conditioner system but had a common above-ceiling plenum with the snack bar/smoking lounge. (Figure 2 locations A and B were part of that study.) At that time construction was underway to build an isolated smoking lounge to the west of the snack bar/smoking lounge by extending new walls from slab to slab, thus eliminating the common above ceiling plenum, and by providing a totally separate air handling system that accomplishes 20 air changes per hour.

In the December 1988 measurements around the computer rooms, spillover levels of 14.1 $\mu g/m^3$ (at A) and 59.0 $\mu g/m^3$ (above the drop ceiling at B) indicated that spillover ETS was encroaching on the computer room. Levels in the computer room were only about 1.1 $\mu g/m^3$.

In September 1989, measurements were carried out in the new isolated smoking lounge and at the point of highest February 1988 readings in the snack bar (No. 3 in Figure 2—178.7 $\mu g/m^3$). These measurements indicated that the snack bar location had decreased to 0.6 $\mu g/m^3$ of nicotine vapor. The new, well-ventilated (20 air changes per hour) and isolated smoking lounge had nicotine vapor levels of 70–77 $\mu g/m^3$.

Along with the above quantitative improvement in the air quality for the snack bar, there was one qualitative observation from the manager of the computer room on the same floor. He reported "a significant decrease in the number of disk crashes" (a problem that had prompted the December

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Table III. Summary of nicotine vapor concentrations averaged by type of exposure on each floor.

| Exposure | Floor | Ave. concn. ($\mu g/m^3$) Before | After | Ratio after/before |
|---|---|---|---|---|
| Smokers | 33nd | 24.0 | 0.4 | 0.02 |
| | 31st | 4.0 | 0.5 | 0.13 |
| | 19th | 4.1 | 0.1 | 0.02 |
| Non-smoker | 32nd | 2.0 | 0.4 | 0.19 |
| | 31st | 1.9 | 0.3 | 0.16 |
| | 19th | 2.2 | 0.1 | 0.02 |
| Vacant | 32nd | 1.6 | 0.1 | 0.06 |
| | 31st | 4.9 | 0.3 | 0.06 |

1988 measurements) and "significantly fewer computer headaches" since the new isolated smoking lounge had gone into use.]

## Conclusions

In general, this environmental tobacco smoke measurement program achieved its objectives and documented various changes in air quality associated with the new policy restricting smoking at one company's headquarters. One can conclude from these measurements that:

- After the policy was implemented, general employee exposure to nicotine vapor (as surrogate for all environmental tobacco smoke) was reduced by over 95 percent on most floors (like the 19th floor). All office areas were reduced to less than 1 $\mu g/m^3$ of nicotine vapor.
- Even the highest concentrations in the general office areas (those adjacent to the designated smoking areas) in February 1988 are much lower than they had been in December 1987.
- While air quality in the work areas of the 31st and 32nd floors improved, the improvement was not as great as on floors that did not share an air handler with a floor having a smoking area. The average level on the 31st floor was four times that on the 19th floor.
- There is evidence that environmental tobacco smoke vapors are spilling over into the office areas of the 32nd floor, probably through open doors and shared overhead plenums.
- Under the new policy the 32nd floor snack bar was measurably quite contaminated with environmental tobacco smoke with worst case levels at least 1,800 times higher than the typical February 1988 office area.
- The cafeteria air quality was, in general, poorer in aver-

Table IV. Comparison of weekly average nicotine vapor concentrations with Wednesday "active" samples (units = $\mu g/m^3$).

| Location # | Phase | Wednesday active | Weekly average[a] | Comments |
|---|---|---|---|---|
| **Offices** | | | | |
| 6 | I | 0.0 | 1.6 | |
| | II | 0.2 | 0.1 | Wed. appears typical |
| 7 | I | 18.7 | 33.3 | Wed. appears typical |
| | II | 0.0 | 0.3 | Wed. appears typical |
| 10 | I | 0.0 | 3.4 | Smoker out M–W |
| | II | 0.2 | 0.7 | Wed. appears typical |
| 22 | I | 0.5 | 4.3 | Smoker in <2.5 hours on Wed. |
| | II | 0.1 | 0.2 | Wed. appears typical |
| 15 | I | 0.1 | 1.9 | |
| | II | 0.0 | 0.3 | Wed. appears typical |
| 24 | I | 0.7 | 1.0 | Wed. appears typical |
| | II | 0.0 | 0.1 | Wed. appears typical |
| **Snack bar** | | | | |
| 3 | I | 7.0 | 10.2 | Wed. appears typical |
| | II | 141.0 | 179.0 | Wed. appears typical |
| **Cafeteria** | | | | |
| 26 | I | 0.4 | 5.0 | Wed. much LOWER |
| | II | 0.3 | 2.4 | Wed. appears LOWER |
| 28 | I | 1.2 | 6.7 | |
| | II | 1.0 | 4.1 | Wed. appears LOWER |

[a] Day only—assumed 45 hr. exposure.

Table V. Summary of Wednesday intensive sampling (units = $\mu g/m^3$).

| Loc. # | Type SNV | Nicotine Vapor Before | After | Change | Particulate Load Before | After |
|---|---|---|---|---|---|---|
| **Office** | | | | | | |
| 7 | S | 18.7 | 0.0 | −18.7 | lost | ND (12) |
| 6 | N | 0.0 | 0.2 | 0.2 | 20 | ND (0) |
| 10 | S → V | 0.0 | 0.2 | 0.2 | 270 | 86 |
| 15 | N | 0.1 | 0.0 | −0.1 | 45 | ND (0) |
| 22 | S → V | 0.5 | 0.1 | −0.4 | lost | ND (12) |
| 24 | N | 0.7 | 0.0 | −0.7 | lost | ND (10) |
| **Snack bar** | | | | | | |
| 3 | | 7.0 | 140.8 | 133.8 | lost | 527 |
| **Cafeteria** | | | | | | |
| 26 | | 0.4 | 0.3 | −0.1 | ND (2) | 32 |
| 28 | | 1.2 | 1.0 | −0.2 | ND (0) | 27 |
| 32 | | | 30.1 | | | 102 |

Notes: lost—sample lost by error.
ND—below detection limit of 20 $\mu g/m^3$.

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age nicotine vapor levels than it was before the new policy restricting smoking went into effect.

- Based on subsequent 1989 measurements, an isolated smoking lounge quantitatively and qualitatively improved conditions elsewhere on the 32nd floor.


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## References


1. S. K. Hammond, B. P. Leaderer, "A diffusion monitor to measure exposure to passive smoking," *Environ. Sci. Technol.* 21: 494 (1987).
2. S. K. Hammond, B. P. Leaderer, A. C. Roche, M. Schenker, "Collection and analysis of nicotine as a marker for environmental tobacco smoke," *Atmos. Environ.* 21: 457 (1987).
3. S. K. Hammond, J. Coghlin, B. P. Leaderer, "Field study of passive smoking exposure with passive sampler," *Proceedings of the 4th International Conference on Indoor Air Quality and Climate*, Vol. 2, 131–136, Institute for Water, Soil and Air Hygiene (Berlin).



William M. Vaughan is with Environmental Solutions, Inc., P.O. Box 11823, St. Louis, MO 63105-0123. S. Katharine Hammond is with the University of Massachusetts Medical School, Worcester, MA 01655.


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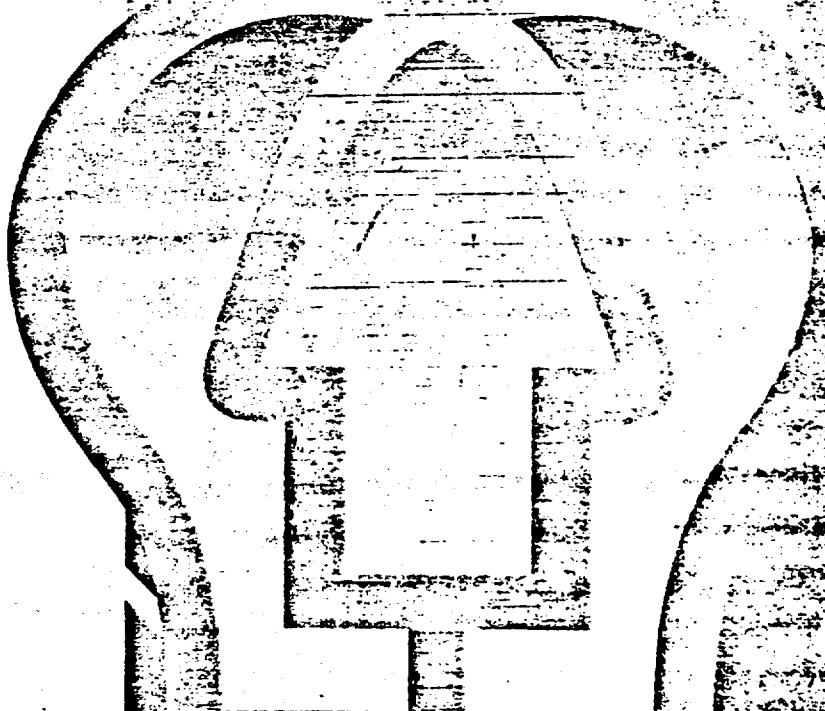

# INDOOR AIR '90

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# BUILDING VENTILATION AND SMOKING POLICY EFFECTS ON INDOOR AIR QUALITY AND EMPLOYEE COMFORT AND HEALTH

Alan Hedge, William A. Erickson
Department of Design and
Environmental Analysis, Cornell University
Ithaca, New York.
U.S.A. 14853

Gail Rubin,
Biometrics Unit, Cornell University
Ithaca, New York.
U.S.A. 14853

Indoor environmental conditions, occupant perceptions of ambient conditions, and reports of "sick" building syndrome (SBS) symptoms were compared for two office buildings: one where smoking is prohibited and one where smoking is restricted to areas with local air cleaning units. Few significant differences in pollutant concentrations were found between buildings. Perceptions of ambient conditions generally were less favorable in the "smoking restricted" building. Apart from mental fatigue, no differences in SBS symptoms were found between these buildings.

## INTRODUCTION

Tobacco smoking in the workplace has been identified as a source of number of indoor air contaminants (1), and it has been reported that passive exposure to environmental tobacco smoke (ETS) may elevate the prevalence of "sick" building syndrome (SBS) symptoms (2). Many organizations have now implemented policies which either prohibit smoking or restrict this in the building, and some companies have also installed local air cleaning systems (LACS) to remove both particulates and volatile organics from air in smoking areas. This study investigated indoor air quality, perceptions of environmental conditions, and health reports in offices in a smoking prohibited (SP) office and a smoking restricted (SR) office with designated smoking areas with LACS.

## MATERIAL & METHODS

Survey Buildings & General Survey Method

Two office buildings occupied by finance/insurance companies were compared. Neither

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building had a history of indoor air quality problems. The SR building is a 40 story, urban office (54,000 $m^3$ gross:1,800 people), with a constant air volume ventilation system. Smoking in the offices was restricted to two smoking lounges with LACS (ceiling mounted units which remove particulate matter and odors via an aluminum pre-filter, ionizer and charcoal filter). The SP building is a four story "greenfield" site office (13,050 $m^3$ gross: 450 people), with a variable air volume ventilation system.

Each building was studied over a two day period in January. On each day two sites were surveyed in the morning and two in the afternoon All sites were in the open plan office areas in the SP building. In the SR building, morning and afternoon measurements were taken in the non-smoking open offices and also in the designated smoking lounges. At the same time a self-report questionnaire, based on previous research instruments for SBS investigations, was distributed at each site. Data on employee perceptions of ambient conditions (16 questions); work-related health and SBS symptoms (17 symptoms); occupational factors (12 questions); ETS exposure and smoking status; and personal information were collected. Answers to the environmental and health questions were made on the same frequency scale, applied to the past month in the building (never; 1-3 times/month; 1-3 times/week; every day). Questionnaire administration and collection were conducted by a researcher on the same day. The return rate was 61% (143/243) in the SP building, and 71% (259/364) in the SP building.

Indoor Air Quality Survey

Soundproofed briefcases containing indoor air quality sampling equipment were unobtrusively placed in each sampling area. One field blank for each sample was taken per building as well as a laboratory method blank per batch. All field and method blanks were found to be uncontaminated. The following pollutants were measured:

a. Nicotine - samples were collected by pumping air at 1 l $min^{-1}$ through sorbent tubes containing XAD-4 resin for 3 hours. Nicotine was not measured in the SP building.
b. Respirable Suspended Particulate (2.5μ cutoff) - samples were taken using a single stage impactor (MSP Corporation) at a 4 l $min^{-1}$ flow rate for 3 hours. Ultra-Violet particulate matter (UV-PM) was analyzed to estimate particulate matter from organic sources (3).
c. Formaldehyde - samples were collected by pumping air a 0.25 l $min^{-1}$ through 2,4 dinitro -phenylhydrazine sorbent tubes for 3 hours (E.P.A. method TO-11).

Hourly measures of the following pollutants were taken with direct reading instruments: carbon monoxide (Interscan 4000); carbon dioxide (Riken 411); suspended particulates (TSI 3500 Piezobalance-3.5μ cutoff); lighting (Minolta Illuminance meter); temperature and relative humidity (Pacer Hygro-thermometer DH300).

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Source: https://www.industrydocuments.ucsf.edu/docs/zqyx0000

Data Analysis

Chi-square analyses of the questionnaire data were performed using SPSSX. The physical environment data first were analyzed using the Generalized Linear Models procedure in SAS. The results reported here are a subset of a larger investigation which is still ongoing. For the SR building, a factorial model of smoking area designation and time-of-day (morning/afternoon) was fit to the environmental data. To test for interactions between buildings and time-of-day, chemical data were analyzed as a split unit design with true replication of time-of-day but no true replication of the building factor. The variation among sites for each building-time combination was used as the error term in testing for an interaction between building and time-of-day; all interactions were tested at a liberal alpha level of 0.1 (4). Independent sample t tests were then used to compare morning and afternoon pollutant concentrations in each building, and to compare pollutant concentrations between buildings separately for morning and afternoon samples. For most of these analyses unequal variance tests were performed.

## RESULTS & DISCUSSION

There was no significant difference between the SP and SR buildings in the percentages of current smokers (17.4% vs. 15.4%), former smokers (12.4% vs. 10.5%), non-smokers (70.3% vs. 74.1%), or in the percentages of managers (13.9% vs. 13.9%), professional workers (18.3% vs. 21.2%), technical workers (16.3% vs. 12.4%), or clerical/secretarial workers (441.8% vs. 47.4%). There was a significant difference between the SP and SR buildings in the percentages of men (17.4% vs. 42.0%) and women (82.6% vs. 58.0%). Analysis of responses to the environmental questions showed that workers in the SR building found thermal conditions over the past month to be colder more often ($X^2 = 19.95$, df=3, $p<0.001$), whereas those in the SP building reported being satisfied with thermal conditions more often ($X^2 = 9.87$, df=3, $p<0.02$). In the SR building there were more complaints of frequent ventilation problems ($X^2 = 11.29$, df=3, $p<0.01$), of too little air movement ($X^2 = 10.48$, df=3 ,$p<0.01$), of dry air ($X^2 = 35.27$, df=3, $p<0.001$), of unpleasant odors ($X^2 = 10.15$, df=3, $p<0.02$), and of stale air ($X^2 = 15.24$, df=3, $p<0.002$) than in the SP building. Proportionally more people in the SR building reported experiencing infrequent glare problems ($X^2 = 12.94$, df=3, $p<0.005$) than in the SP building.

Health symptoms were defined as work-related only if the respondent indicated that the symptom got better on leaving the building. There were no significant differences between the SP and SR buildings in any work-related symptoms (table 1), except for "mental fatigue" ($X^2 = 14.22$, df=3, $p<0.003$).

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Source: https://www.industrydocuments.ucsf.edu/docs/zqyx0000

The indoor environment data are summarized in tables 2 and 3. Data for carbon monoxide, carbon dioxide,and formaldehyde were transformed (natural log) because these data appeared to be lognormal (the variance varied with the mean). For computational purposes a value of $10^{-4}$ was added to each carbon monoxide reading since there were zero readings. There were no significant differences between buildings for morning samples for any pollutant. There was no significant difference in carbon dioxide and UV-PM between buildings for the afternoon samples. Afternoon concentrations of carbon monoxide (equal variance $t = 3.34$, $df=4$, $p<0.03$) and formaldehyde ($t = 4.37$, $df=3.8$, $p<0.01$) were higher in the SR building. Comparisons between morning and afternoon samples for carbon monoxide, carbon dioxide, formaldehyde, and UV-PM were not significant within either building. Analyses of the hourly data for particulates, air temperature, relative humidity, and illumination showed no significant differences between morning and afternoon conditions in the SP building. There were no morning/afternoon differences in particulates, relative humidity, or illumination for the SR building; however, air temperature was significantly higher in the afternoon in this building ($t = 15.81$, $df=1.4$, $p<0.03$ ).

Comparisons between buildings showed no morning differences, except for relative humidity ($t = 5.04$, $df=3.7$, $p<0.01$). In the afternoon, there were no differences between the buildings for carbon dioxide, or particulates; but there were significant differences for formaldehyde ($t = 4.37$, $df=3.8$, $p<0.01$), air temperature ($t = 4.30$, $df=3.2$, $p<0.02$), relative humidity ($t = 2.90$, $df=3.6$, $p<0.05$), illumination ($t = 6.03$, $df=1.8$, $p<0.05$), and UV-PM (equal variance $t = 29.40$, $df=1$, $p<0.02$).

In the SR building, there were no significant differences in pollutant concentrations between morning and afternoon samples for the designated smoking areas. Comparisons of designated smoking and open-office areas showed no morning or afternoon differences in the concentrations of carbon monoxide, carbon dioxide, air temperature, relative humidity, or illumination. However, morning data showed a significant difference in concentrations of particulates ($t = 11.8$, $df=1.1$, $p<0.05$), and marginally significant differences in concentrations of UV-PM ($t = 6.31$, $df=1.0$, $p<0.1$), and nicotine ($t = 8.15$, $df=1.1$, $p<0.07$). For the afternoon samples, these comparisons showed no significant differences in concentrations of particulates or UV-PM; but there were marginally significant differences in concentrations of formaldehyde ($t = 6.58$, $df=2.0$, $p<0.07$) and nicotine ($t = 8.08$, $df=1.0$, $p<0.08$).

## CONCLUSIONS

Perceptions of environmental conditions were generally less favorable for the SR building than the SP building. Pollutant concentrations for carbon monoxide, carbon dioxide, formaldehyde, and UV-PM were significantly higher for afternoon samples in the SR than in

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Source: https://www.industrydocuments.ucsf.edu/docs/zqyx0000

Nicotine also was measurable in the SR building. However, except for mental fatigue, there were no significant differences in the prevalence of work-related SBS symptoms between buildings. These results suggest that restricting smoking to designated areas, which have local air filtration systems, is effective in minimizing the impact of environmental tobacco smoke on indoor air quality, and that SBS is not caused by exposure to environmental tobacco smoke in offices.

## ACKNOWLEDGEMENTS

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## REFERENCES

1. Hodgson, M.J. (1989) The role of combustion products in building-associated illness. Occupational Medicine: State of the Art Reviews, 4, 735-740.
2. Robertson, A.S., Burge, P.S., Hedge, A. et al (1988) Relation between passive cigarette smoke exposure and "building sickness". Thorax, 43, 263.
3. Ingebrephsen, B.J. et al. (1988) A comparative study of ETS particulate mass measurements in an environmental chamber. Journal of the Air Pollution Control Association, 38, 413-417.
4. Snedecor, G.W. and Cochran, W.G. (1980) Statistical Methods (7th ed), Iowa State University Press.

Table 1 The prevalence of SBS symptoms experienced at least once in the past month.

| SBS symptoms | SP building | SR building |
|---|---|---|
| Dry eyes | 39.0% | 43.9% |
| Sore eyes | 40.1% | 48.6% |
| Sore throat | 23.6% | 26.2% |
| Stuffy nose | 32.4% | 32.9% |
| Runny nose | 25.2% | 24.5% |
| Headache | 49.4% | 44.4% |
| Chest tightness | 8.9% | 10.7% |
| Lethargy | 31.7% | 33.8% |
| Mental fatigue | 57.5% | 41.5% p<0.002 |

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Source: https://www.industrydocuments.ucsf.edu/docs/zqyx0000

Table 2 Concentrations of carbon monoxide, carbon dioxide, and formaldehyde in the offices. (mean ppm value is the mean back transformed from the log)

| | | AM samples | | | PM samples | | |
|---|---|---|---|---|---|---|---|
| | | Mean | | Mean | Mean | | Mean |
| Pollutant | Building | log | SE | ppm | log | SE | ppm |
| Carbon monoxide | SP | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 |
| | SR (Office) | 0.9 | 0.3 | 2.5 | 0.5 | 0.3 | 1.7 |
| | SR (Smoking) | 1.0 | 0.3 | 2.8 | 1.0 | 0.3 | 2.6 |
| Carbon dioxide | SP | 6.38 | 0.04 | 589 | 6.41 | 0.04 | 610 |
| | SR (Office) | 6.73 | 0.12 | 845 | 6.46 | 0.04 | 642 |
| | SR (Smoking) | 6.53 | 0.00 | 684 | 6.58 | 0.07 | 717 |
| Formaldehyde | SP | -2.91 | 0.14 | 0.023 | -3.06 | 0.07 | 0.019 |
| | SR (Office) | -3.78 | 0.52 | 0.008 | -3.39 | 0.03 | 0.012 |
| | SR (Smoking) | -3.09 | 0.08 | 0.018 | -2.88 | 0.07 | 0.023 |

Table 3 Concentrations of ETS pollutants and ambient conditions in the offices.

| | | AM samples | | PM samples | |
|---|---|---|---|---|---|
| Pollutant | Building | Mean | SE | Mean | SE |
| Nicotine ($\mu g\ m^{-3}$) | SR (Office) | 0.0 | 0.0 | 0.0 | 0.0 |
| | SR (Smoking) | 27.2 | 3.2 | 41.2 | 5.0 |
| Particulates: | SP | 20 | 0.0 | 10 | 10 |
| ($3.5\mu$ - $\mu g\ m^{-3}$) | SR (Office) | 40 | 10 | 40 | 10 |
| | SR (Smoking) | 110 | 0.0 | 140 | 20 |
| Particulates: | SP | N/A | N/A | N/A | N/A |
| ($2.5\mu$ - $\mu g\ m^{-3}$) | SR (Office) | 200.0 | 0.0 | 300.0 | 100.0 |
| | SR (Smoking) | 350.0 | 50.0 | 400.0 | 0.0 |
| UVPM ($\mu g\ m^{-3}$) | SP | 0.0 | 0.0 | 0.0 | 0.0 |
| | SR (Office) | 9.0 | 1.0 | 7.0 | 0.0 |
| | SR (Smoking) | 120.0 | 18.0 | 185.0 | 52.0 |
| Temp. (°C) | SP | 24.0 | 0.1 | 23.5 | 0.3 |
| | SR (Office) | 23.7 | 0.0 | 24.6 | 0.1 |
| | SR (Smoking) | 23.0 | 0.5 | 24.2 | 0.2 |
| Relative Humidity (%) | SP | 36.5 | 2.3 | 35.5 | 4.6 |
| | SR (Office) | 21.9 | 1.8 | 21.4 | 1.5 |
| | SR (Smoking) | 22.4 | 0.7 | 21.7 | 1.8 |
| Illumination (lux) | SP | 388 | 100 | 304 | 34 |
| | SR (Office) | 425 | 148 | 712 | 58 |
| | SR (Smoking) | 664 | 102 | 456 | 344 |

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Source: https://www.industrydocuments.ucsf.edu/docs/zqyx0000

# ETS in Offices and When Smoking Is Restricted to Designated But Not Separately Ventilated Areas*

T. D. Sterling and B. Mueller

## Summary

Nicotine, respirable suspended particles (RSP), carbon monoxide (CO), and carbon dioxide ($CO_2$) levels were measured in the smoking and nonsmoking sections of two cafeterias, a smoking lounge, and several offices.

Smoking in the offices was regulated by one of three methods:

1) smoking permitted *ad lib*,
2) smoking prohibited except in designated areas which were not separately ventilated, and
3) smoking prohibited except in designated areas which were separately ventilated.

Nicotine levels in the nonsmoking offices which received recirculated air from a designated smoking area were less than 1.0 µg/m³. There was no difference in average RSP, CO, and $CO_2$ concentrations between nonsmoking offices that received recirculated air from designated smoking areas and nonsmoking offices that did not receive recirulated air. The results indicate that the provision of a designated, but not separately ventilated smoking area can effectively eliminate or drastically reduce most components of environmental tobacco smoke from non-smoking offices.

## Introduction

Policies to regulate smoking in offices have been developed and implemented by private companies and some government agencies in response to concerns of health effects from involuntary exposure to environmental tobacco smoke (ETS). Several North American municipalities have passed bylaws to regulate smoking in all public buildings and the Canadian and American federal governments are currently developing methods to regulate smoking in workplaces under their jurisdiction. Most of the current or proposed bylaws establish no smoking as the workplace norm, except for specially designated areas where smoking is permitted.

Four options are available to regulate smoking in the office-work area:

1) Outright prohibition of smoking in all building locations;
2) Restricting smoking to designated areas that are separately ventilated;
3) Restricting smoking to designated areas that are not separately ventilated;
4) Providing some framework by which an adjustment between smoking and non-smoking workers can be achieved, without directly regulating the placement of smokers.

The third option, that of providing a designated but not separately ventilated smoking area, appears to be the most frequently adopted procedure. The first option, an outright ban on smoking throughout a building, is usually an unsatisfactory solution because a certain proportion of building occupants are smokers who demand a location where they can be allowed to smoke. Smokers are found not only among employees but also among members of the public who are waiting for services. Buildings such as prisons or hospitals have full-time residents, some of whom are smokers. The second option, restricting smoking to designated areas that are separately ventilated, is often not a viable alternative because installing separate ventilation in an existing building may be physically impossible or very expensive. The fourth option is infrequently adopted because of objections by non-smokers or because of existing municipal regulations. Thus, the least disruptive and least costly solution for many buildings is to restrict smoking to designated, but not separately ventilated, areas. However, it is necessary to determine if the third option can reduce ETS constituents to acceptable levels.

This report presents the results of two separate field studies of ETS levels in Canadian offices under conditions of normal occupancy, smoking and ventilation.

The two studies were designed to provide data on two questions:

1) What is the contribution of smoking to ambient levels of four substances: $CO_2$, CO, nicotine, and RSP in offices?
2) How does restricting smoking to specially designated, but not separately ventilated areas, affect the levels of these four substances in nonsmoking offices?

## Methods

### *Series I*

In the first study, the number of office occupants and number of cigarettes smoked were observed while ambient levels of nicotine, CO and $CO_2$ were measured on the 7th and 11th floors of a government office building. (Measurements of RSP were also taken but, unfortunately, turned out to be inappropriately analyzed.) Smoking was permitted *ad lib* on the 11th floor but was restricted on the 7th floor to a 22.5 m² (242 ft²) coffee/smoking lounge where smoking was permitted at all times. The layout and design of the two floors were almost identical, with an open-area office of approximately 780 m² (8,400 ft²) surrounding a 114 m² (1,230 ft²) mechanical/service core.

Each floor had it's own independent ventilation system which recirculated between 80% to 85% of the air returned from each floor and provided at least 20 cfm (cubic feet per minute) of fresh air per person. Except for leakage through elevator shafts and stairwells, no mixing or recirculation of air between floors occurred. The coffee/smoking lounge on the 7th floor was on the same ventilation system as the rest of the 7th floor.

* Part of the costs of this project came from a special grant from the council for Tobacco Research Inc.

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### *Series II*

In the second study, the number of office occupants and cigarettes smoked were observed while ambient levels of RSP, nicotine, CO and $CO_2$ were measured in two adjacent buildings (A and B) containing a mixture of open-area offices, private offices and public waiting/service areas.

Building A was a sealed, mechanically ventilated four storey office building with two levels of underground parking. Each of the four floors contained approximately 1,390 $m^2$ (15,000 $ft^2$) of office space. Smoking was prohibited in all areas of the building except for a smoking section, in the fourth floor cafeteria, which was not separately ventilated. The ventilation system mixed indoor air from all parts of the building before recirculation.

Building B was a 12-storey unsealed office building where most areas were passively ventilated by building leakage. Few areas had a separate ventilation system, and these systems were not connected to other ventilation systems. Consequently, there was no mechanical mixing of air from different floors or offices. Smoking was prohibited in all areas of the building except in the smoking section of a basement cafeteria. Heated/cooled air was supplied separately to the cafeteria and exhausted through windows.

### *Sampling Methods*

**Nicotine:** In Series I, eight 1-h nicotine samples were collected in the designated smoking room, ten 1-h samples in the non-smoking offices on the 7th floor, and ten 1-h samples in the smoking-permitted offices on the 11th floor. In Series II, six 1-h nicotine samples were collected in each of the smoking and non-smoking sections of the cafeterias of Buildings A and B; two samples on each of the four floors in the non-smoking offices of Building A and two samples in non-smoking offices of Building B. Of the samples obtained in the non-smoking offices of buildings A and B, six collected air for 2 h, three collected air for 4 h and one collected air for 8 h.

Ambient nicotine was sampled with a portable air sampling pump housed inside a briefcase. The sampling apparatus was designed to collect samples unobtrusively because of the previously noted effect of observation on occupant behavior (Health and Welfare Canada 1985a). Nicotine samples were collected by pumping air at 1 l/min through sorbent tubes containing XAD-4 resin, a styrene divinylbenzene copolymer. The sorbent tubes contained 80 mg of resin in the front (primary) section and 40 mg in the rear (secondary) section. After sampling, the sorbent tubes were refrigerated until analysis.

The analytical procedure for nicotine was based on NIOSH (1977). Resin beads from the primary section of sorbent tubes were transferred to gas chromatograph autosampler vials containing 1 ml of ethyl acetate as the extraction solvent, 5 mg/l quinoline as an internal standard and 0.01% by volume of triethylamine to prevent adsorptive losses of nicotine onto the glass autosampler vials. Samples and spiked standards were placed on an automatic shaking device and shaken for 30 min. A Hewlett-Packard Model 5880A or Model 5830A gas chromatograph equipped with a nitrogen-phosphorous detector, an autosampler and a GC terminal were used to determine peak areas of the nicotine samples and standards. The assayed nicotine was corrected for the desorption efficiency (usually 94%) of the particular lot of XAD-4 resin used in sampling. The final weight of nicotine detected was divided by the volume of air sampled to give results in $\mu g/m^3$. The rear (backup) sections of sorbent tubes were analyzed separately and, except for one case, gave nicotine determinations less than the detection limit, indicating no break-through of nicotine past the primary resin section.

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**RSP:** RSPs were measured in Series II during the entire period of nicotine sampling and were averaged over each sampling period. RSP (particles less than 5 μm diameter) levels were measured with a Sibata Scientific Technology P-5H digital dust indicator which senses light side-scattered by suspended particles. The unit was calibrated at the factory to monodispersed stearic acid particles with a mean diameter of 0.3 μm. The digital counts of particles per sampling time were converted to RSP levels in $\mu g/m^3$.

**CO and $CO_2$:** Co and $CO_2$ levels were measured over 3–4-min-periods approximately midway into the 1- or 2-h nicotine sampling periods and at least twice during the 4- or 8-h nicotine sampling periods. CO was measured using a direct reading electrochemical analyzer (Nova 310L) housed in a flight-case. $CO_2$ was measured using extra low range $CO_2$ Gastec detector tubes and a manual sampling pump.

**Other Observations:** During each sampling period, the number of occupants and the number of cigarettes smoked in each predefined observation area were recorded. The observation areas were defined by the ability to survey the area. For purpopses of comparison, the average number of persons per 10 $m^2$ and cigarettes smoked per hour per 10 $m^2$ were calculated.

## Results

### *Series I*

Table 1 presents the average concentrations of nicotine, CO and $CO_2$, the average number of persons per 10 $m^2$, and the number of cigarettes smoked per hour per 10 $m^2$ (where applicable) for the smoking-permitted and non-smoking floors and the designated smoking area.

Although the three areas differed substantially in the number of cigarettes smoked per hour per 10 $m^2$, only ambient nicotine levels responded in a similar fashion. For example, the average smoking intensity (cigarettes per hour per 10 $m^2$) was 10.8 times greater in the smoking designated area than in the smoking-permitted floor. Similarly, the average ambient nicotine level was 15.6 times greater in the smoking designated than the smoking-permitted areas. Corresponding ratios for $CO_2$ and CO were only 1.3 and 1.7 respectively.

**Table 1.** Comparison of ETS related air quality variables (averages) in smoking prohibited and permitted work areas and in designated smoking areas, site 1

| | Nicotine ($\mu g/m^3$) | CO (ppm) | $CO_2$ (ppm) | Persons per 10 $m^2$ | Cigarettes/ h/10 $m^2$ |
|---|---|---|---|---|---|
| Smoking permitted | 4.8 | 2.5 | 720 | 0.79 | 0.36 |
| Smoking prohibited | $<1.6$ | 2.1 | 680 | 0.61 | NA |
| Designated smoking | 75 | 4.2 | 960 | 0.97 | 3.9 |

Source: https://www.industrydocuments.ucsf.edu/docs/zqyx0000

Smoking was not observed on the non-smoking floor. The average ambient nicotine concentrations were below the limit of detection (i.e. less than 1.6 μg/m³) for the 1-h sampling periods used. CO and $CO_2$ levels were slightly lower on the non-smoking floor than on the smoking-permitted floor.

Table 1 also lists the average person densities for each of the three sampling areas. The average person density was 30% higher on the smoking-permitted floor and 60% higher in designated smoking area than on the non-smoking floor. Corresponding ratios calculated for CO and $CO_2$ show a similar pattern and range.

## *Series II*

The data for the smoking and non-smoking cafeteria sections of Buildings A and B were combined because there were only very small differences in the results for each building.

Figs. 1 through 4 summarize the ambient $CO_2$, CO, RSP and nicotine concentrations, in four areas under different smoking and ventilation conditions: the smoking sections of the two cafeterias, the non-smoking sections of the two cafeterias, the non-smoking offices of Building A which received recirculated air from other areas of the building, and the separately ventilated non-smoking offices of Building B which did not receive recirculated air from other areas of the building. In each figure, the height of the bars give the average concentration, n gives the total number of samples on which the average is based. The range of observed values is given by the vertical line.

Figure 1 shows that there was little difference between the $CO_2$ levels in the smoking prohibited offices of building A and building B, whether or not they received recirculated air. The smoking and non-smoking sections of the cafeterias had slightly elevated, but not statistically significant, $CO_2$ concentrations when compared to the non-smoking offices. These small increases in $CO_2$ levels could have been partly due to occupant density which was twice as high in the cafeteria sections than in the non-smoking offices.

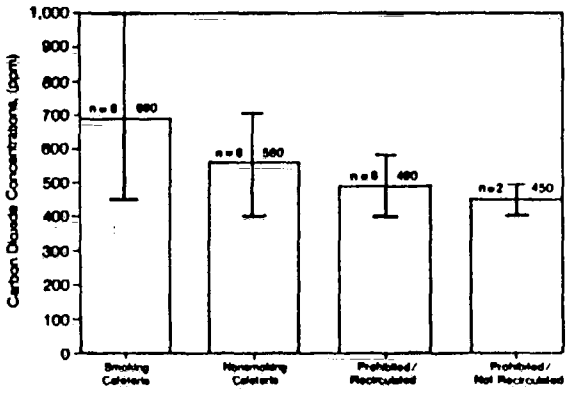


Fig. 1. Comparison of averages and ranges of $CO_2$ concentrations for different smoking regulations, series II

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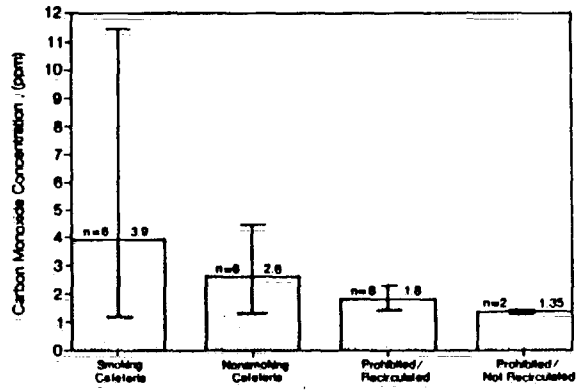


Fig. 2. Comparison of averages and ranges of CO concentrations for different smoking regulations, series II

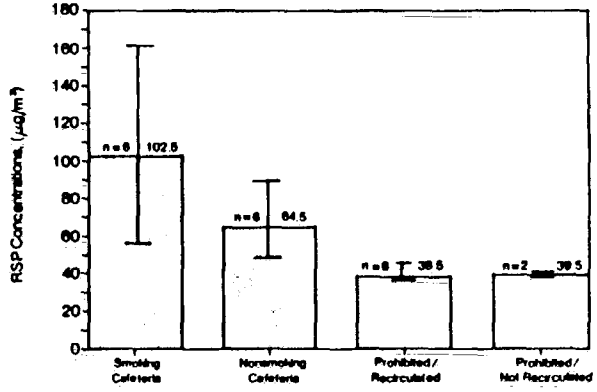


Fig. 3. Comparison of averages and ranges of RSP concentrations for different smoking regulations, series II

The distribution of mean ambient CO levels is similar to that for $CO_2$, as shown in Fig. 2. Although mean CO levels were higher in both cafeteria sections than in the non-smoking offices, none of the differences were statistically significant.

There were no differences in RSP concentrations between the non-smoking offices with and without recirculated air, as shown in Fig. 3. However, the mean RSP level in the smoking section of the cafeterias was 2.6 times that in the non-smoking offices while the mean level in the non-smoking section of the cafeterias was about 1.7 times that found in non-smoking offices.

Source: https://www.industrydocuments.ucsf.edu/docs/zqyx0000

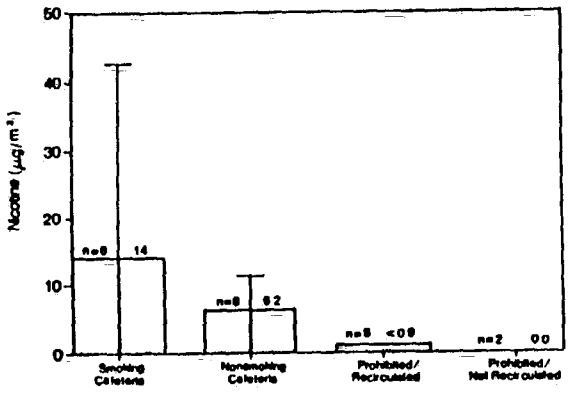


**Fig. 4. Comparison of averages and ranges of nicotine concentrations for different smoking regulations**

Figure 4 gives average ambient nicotine concentrations. In some instances the nicotine levels in the cafeteria sections were below the detection limit of 1.6 μg/m³. The mean nicotine concentration measured in the smoking sections of the cafeterias was more than twice that in the non-smoking sections of the cafeterias and at least 15 times that in the non-smoking offices which received recirculated air.

The lower detection limit for nicotine is dependent upon the amount of air that is sampled. For the method used in this study, the lower detection limit was 0.8 μg/m³ for a 2-h sample, 0.4 μg/m³ for a 4-h sample, and 0.2 μg/m³ for an 8-h sample. Two-, four- and eight-hour samples were taken in non-smoking offices in Building A which received recirculated air. None of four 2-h samples were above the detection limit; one of three 4-h samples was above the detection limit and gave a determination of 1.0 μg/m³; the single 8-h sample was also above the detection limit and yielded a determination of 0.8 μg/m³. These results indicate that the ambient nicotine concentration in these non-smoking offices was not larger than the maximum positive result of 1.0 μg/m³.

Two 2-h nicotine samples were taken in non-smoking offices in building B which did not receive recirculated air. Nicotine was below the detection limit of 0.8 μg/m³ in both samples.

## Discussion

The Series I results suggest that ETS contributes little to ambient $CO_2$ levels. The differences in $CO_2$ concentrations between the smoking-permitted, prohibited and designated smoking areas were small compared with the observed differences in smoking intensity (cigarettes smoked per hour per 10 m²) and ambient nicotine levels. However, the sample sites with higher $CO_2$ levels in Series I and II also had higher person densities. This suggests that people were the primary source of $CO_2$.

Ambient CO concentrations increased with smoking (Table 1 and Fig. 2) but did not closely follow smoking intensity or nicotine concentrations. Other indoor and outdoor

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sources of CO must also contribute to CO levels, as indicated by the background level of 1.35 ppm in the non-smoking office without air recirculation. Part of the higher CO level in the cafeteria could also be due to cooking activities.

RSP are produced both by smoking and by many other processes. The background RSP level, as indicated by the results for the non-smoking office without recirculated air, are about 39 μg/m³.

Not surprisingly, of the four substances measured, nicotine shows the strongest association with smoking. There are few if any significant sources of nicotine in the non-industrial indoor environment other than smoking and it follows that nicotine is an accurate marker of ETS exposure. Improvements in nicotine measurement technology could result in the widespread use of nicotine as an indicator of ETS exposure (HWC 1987).

Restricting smoking to specially designated areas which are not separately ventilated appears to effectively prevent high ETS levels in adjacent non-smoking areas. Both RSP and nicotine concentrations declined sharply from the smoking to the non-smoking sections of the cafeterias in Buildings A and B (Figs. 3 and 4). The recirculation of air from the smoking and non-smoking sections of the Building A cafeteria further diluted ETS to the extent that the levels of CO, $CO_2$, and RSP in the non-smoking offices of Building A were approximately the same as those levels in the non-smoking office in building B, which did not receive recirculated air.

Nicotine levels were at or below 1 μg/m³ in non-smoking offices which receive recirculated air from smoking designated areas. This level of exposure is very low. For example, breathing air which contains 1.0 μg/m³ of nicotine for 1 h at an average respiration rate for office activity of 0.48 m³/h (ASHRAE 1986) is approx. equal to 1/1,900th of the 900 μg of nicotine inhaled by a smoker from the mainstream smoke of one cigarette (Muramatsu et al. 1984).

There are few published studies of nicotine levels in non-smoking offices. The nicotine levels observed by Bayer and Black (1986) are, unfortunately, not comparable with the results given here because their results are given in ng/m²/min. Nevertheless, they did not detect nicotine in two of three non-smoking offices.

Other office studies have also found low RSP levels in non-smoking areas close to smoking designated areas that are not separately ventilated (Lee 1985). These findings are reinforced by Health and Welfare Canada (HWC 1985a) who measured RSP, CO and $CO_2$ concentrations before and after the implementation of a no smoking policy in offices that received recirculated air from a designated smoking area. After the no smoking policy was implemented, mean RSP levels decreased by 8 μg/m³, from 26 μg/m³ to 18 μg/m³ (HWC 1985a).

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The results of the Series II and the HWC (1985a) studies indicate that recirculated air from designated smoking areas contributes less than 8 μg/m³ of RSP to non-smoking offices which receive such air. HWC (1985b) further suggests that *ad lib* smoking in offices under normal ventilation and occupancy conditions contributes about 7 μg/m³. These findings are substantially different from the widely quoted estimate by Repace and Lowrey (1980) that smoking increases RSP levels in offices by 170 to 200 μg/m³. This

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Table 2. Comparison of RSP mean concentrations on office floors before and after a no smoking policy was implemented (extracted from HWC, August, 1985)

| | RSP ($\mu g/m^3$) | $CO_2$ (ppm) |
|---|---|---|
| Before no smoking policy | | |
| Floor A | 29.3 | 663 |
| Floor B | 30.0 | 614 |
| Floor C | 25.0 | 606 |
| Overall average | 28.1 | 627 |
| After no smoking policy | | |
| Floor A | 22.7 | 591 |
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While smoking regulations are here to stay and will affect most offices under federal, provincial or municipal control in Canada, the haste to regulate smoking may have been based on the unrealistic modelled estimates of Repace and Lowrey or on "worst case" measurements in poorly ventilated workplaces, instead of on actual measurements of RSP levels in typical offices.

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**Acknowledgements:** We thank Mr. C. Collett for his help in arranging for sampling sites. We are beholden to Dr. C. Nystrom for help with some of the equipment used for Series 1.

*References*

ASHRAE (1986) ASHRAE Standard 62-1981R, Ventilation for acceptable indoor air quality. American Society of Heating, Refrigerating and Air-Conditioning Engineers, Atlanta, Georgia

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Bayer CW, Black MS (1986) Passive smoking: Survey analysis of office smoking areas vs. environmental chamber studies. Atlanta, Georgia (Proceedings, ASHRAE IAQ '86, pp 281–291)

Health and Welfare Canada (1985a) Investigation on the impact of a new smoking policy on office air quality. National Library of Canada Hull, Quebec. Occupational Health Unit Medical Services Branch

Health and Welfare Canada (1985b) Investigation on the impact of a new smoking policy on office air quality. Office of the Auditor General Ottawa, Ontario. Occupational Health Unit Medical Services Branch

Health and Welfare Canada (1987) Involuntary exposure to tobacco smoke. Report of the Advisory Committee, Environmental Health Directorate, Ottawa

Lee HK (1985) Investigation on the impact of a new smoking policy on office air quality in the National Library of Canada in Hull, Quebec. Ottawa (Proceedings of the 78th Annual Meeting of the Air Pollution Control Association)

Muramatsu M, Umemura S, Okada T, Tomita H (1984) Estimation of personal exposure to tobacco smoke with a newly developed nicotine person monitor. Environ Res 35:218–227

NIOSH (1977) Manual of analytical methods, 2nd ed. US National Institute of Occupational Safety and Health, Cincinnati, Ohio

Repace JL, Lowrey AH (1980) Tobacco smoke, ventilation, and indoor health. Science 208:464–472

Sterling TD, Collett CW, Sterling EM (1987) Environmental tobacco smoke and indoor air quality in modern office work environments. J Occup Med 29:57–62

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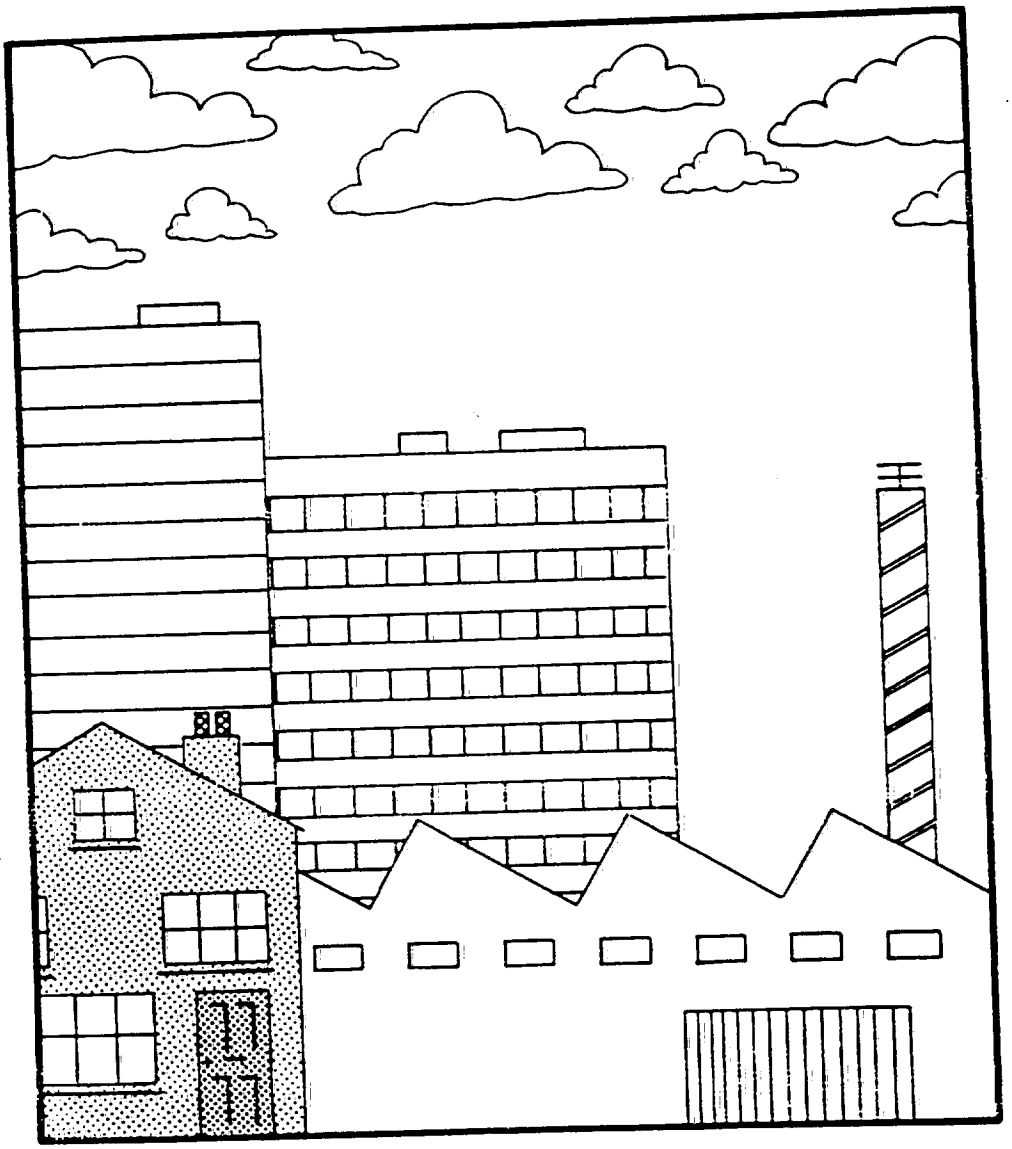
# INDOOR AND AMBIENT AIR QUALITY



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and P.W. Kirk

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# INDOOR AND AMBIENT AIR QUALITY

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# ETS CONCENTRATIONS UNDER DIFFERENT CONDITIONS OF VENTILATION AND SMOKING REGULATION

T.Sterling

Faculty of Applied Sciences, Simon Fraser University, Burnaby, B.C. V5A 1S6, Canada

ABSTRACT

Concentrations of nicotine, respirable particulates (RSP), carbon monoxide (CO) and carbon dioxide ($CO_2$) were measured in offices under conditions of: smoking ad lib; smoking prohibited; smoking prohibited but receiving recirculated air from designated smoking areas that are not separately ventilated; smoking prohibited and smoking in designated areas that are separately ventilated.

## INTRODUCTION

Smoking is being rapidly regulated in public buildings especially in offices. The source of regulation usually is management, acting sometimes under pressure by non smoking employees. However, an increasing number of municipalities are passing bylaws setting the framework for smoking regulation in offices. Departments of the federal government, and units within these departments may develop their own smoking policies. The Canadian Federal Government is in the process of developing a general smoking policy for all its employees. Other areas than those serving as offices may also fall under smoking regulations. For instance, a number of Canadian provincial and federal courts have set aside non smoking jury rooms.

Smoking regulations may be of four basic types. Smoking may be prohibited entirely; or to the contrary, smoking may be permitted in the working area but under some local arrangements by which smokers and non smokers apportion the workspace among each other. In most instances, smoking may be restricted to a designated area, typically a cafeteria or a room usually serving as a coffee or lunch room.

Of these three options, the third will affect most office and public buildings.

In some buildings, the smoking designated area may be separately ventilated so that no component of Environmental Tobacco Smoke (ETS) may mix with the air circulating through the non smoking working areas. However, in many and possibly the majority of existing buildings, smoking will be designated to an area that is physically separate from the work area but is not separately ventilated. Components of ETS from such areas could mix with the recirculated air penetrate non smoking areas but in some diluted form.

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Because of the different types of interaction between a building's ventilation and location of its smoking areas, a number of questions concerning air quality arise. Of these, two questions are of special interest. The first concerns the contribution of smoking to ambient levels of substances related to ETS, most importantly to nicotine, CO, $CO_2$, and respirable particulates (RSP) and the second asks just how does restricting smoking to specially designated but not separately ventilated areas affect the levels of ETS related substances in non smoking offices?

This report presents the results of two separate field studies of ETS levels in Canadian offices under conditions of normal occupancy, smoking and ventilation, and where in some cases designated smoking areas were, and in some cases were not, separately ventilated.

## METHODS

### FIELD STUDY I

In the first study, the number of office occupants and number of cigarettes smoked were observed while ambient levels of nicotine, CO and $CO_2$ were measured on the 7th and 11th floors of a government office building. Smoking was permitted *ad lib* on the 11th floor but was restricted on the 7th floor to a 22.5 $m^2$ (242 $ft^2$) coffee/smoking lounge where smoking was permitted at all times. The layout and design of the two floors were almost identical, with an open-area office of approximately 780 $m^2$ (8,400 $ft^2$) surrounding a 114 $m^2$ (1,230 $ft^2$) mechanical/service core.

Each floor had it's own independent ventilation system which recirculated between 80% to 85% of the air returned from each floor and provided at least 20 cfm (cubic feet per minute) of fresh air per person. Except for leakage through elevator shafts and stairwells, no mixing or recirculation of air between floors occurred. The coffee/smoking lounge on the 7th floor was on the same ventilation system as the rest of the 7th floor.

### FIELD STUDY II

In the second study, the number of office occupants and cigarettes smoked were observed while ambient levels of RSP, nicotine, CO and $CO_2$ were measured in two adjacent buildings (A and B) containing a mixture of open-area offices, private offices and public waiting/service areas.

Building A was a sealed, mechanically ventilated four storey office building with two levels of underground parking. Each of the four floors contained approximately 1,390 $m^2$ (15,000 $ft^2$) of office space. Smoking was prohibited in all areas of the building except for a smoking section, in the fourth floor cafeteria, which was not separately ventilated. The ventilation system mixed indoor air from all parts of the building before recirculation.

Building B was a 12-storey unsealed office building where most areas were passively ventilated by building leakage. Few areas had a separate ventilation system, and these systems were not connected to other ventilation systems. Consequently, there was no mechanical mixing of air from different floors or offices. Smoking was prohibited in all areas of the building

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except in the smoking section of a basement cafeteria. Heated/cooled air was supplied separately to the cafeteria and exhausted through windows.

Sampling Methods

Nicotine:

In FIELD STUDY I, eight one-hour nicotine samples were collected in the designated smoking room, ten one-hour samples in the nonsmoking offices on the 7th floor, and ten one-hour samples in the smoking-permitted offices on the 11th floor. In FIELD STUDY II, six one-hour nicotine samples were collected in each of the smoking and nonsmoking sections of the cafeterias of Buildings A and B; two samples on each of the four floors in the nonsmoking offices of Building A and two samples in nonsmoking offices of Building B. Of the samples obtained in the nonsmoking offices of buildings A and B, six collected air for two hours, three collected air for four hours and one collected air for eight hours.

Ambient nicotine was sampled with a portable air sampling pump housed inside a briefcase. The sampling apparatus was designed to collect samples unobtrusively because of the previously noted effect of observation on occupant behavior (1). Nicotine samples were collected by pumping air at 1 litre per minute through sorbent tubes containing XAD-4 resin, a styrene divinylbenzene copolymer. The sorbent tubes contained 80 mg of resin in the front (primary) section and 40 mg in the rear (secondary) section. After sampling, the sorbent tubes were refrigerated until analysis.

The analytical procedure for nicotine was based on NIOSH (2). (Further detail of this procedure is given in Sterling (3)).

RSP:

RSP's were measured in FIELD STUDY II during the entire period of nicotine sampling and were averaged over each sampling period. RSP (particles less than 5 $\mu$m diameter) levels were measured with a Sibata Scientific Technology P-5H digital dust indicator which senses light side-scattered by suspended particles. The unit was calibrated at the factory to monodispersed stearic acid particles with a mean diameter of 0.3 $\mu$m. The digital counts of particles per sampling time were converted to RSP levels in $\mu g/m^3$.

CO and $CO_2$:

CO and $CO_2$ levels were measured over three to four minute periods approximately midway into the one- or two- hour nicotine sampling periods and at least twice during the four- and eight-hour nicotine sampling periods. CO was measured using a direct reading electrochemical analyzer (Nova 310L) housed in a flight-case. $CO_2$ was measured using extra low range $CO_2$ Gastec detector tubes and a manual sampling pump.

Other Observations:

During each sampling period, the number of occupants and the number of cigarettes smoked in each predefined observation area were recorded. The observation areas were defined by the ability to survey the area. For

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purposes of comparison, the average number of persons per 10 $m^2$ and cigarettes smoked per hour per 10 $m^2$ were calculated.

## RESULTS

### FIELD STUDY I

Table 1 presents the average concentrations of nicotine, CO and $CO_2$, the average number of persons per 10 $m^2$, and the number of cigarettes smoked per hour per 10 $m^2$ (where applicable) for the smoking-permitted and nonsmoking floors and the designated smoking area.

TABLE 1

Comparison of ETS Related Air Quality Variables (Averages) in Smoking Prohibited and Permitted Work Areas and in Designated Smoking Areas
FIELD STUDY I

| | Nicotine ($\mu g/m^3$) | CO (ppm) | $CO_2$ (ppm) | Persons per $10m^2$ | Cigarettes/ hour/$10m^2$ |
|---|---|---|---|---|---|
| Smoking Permitted | 4.8 | 2.5 | 720 | 0.79 | 0.36 |
| Smoking Prohibited | <1.6 | 2.1 | 680 | 0.61 | NA |
| Designated Smoking | 75 | 4.2 | 960 | 0.97 | 3.90 |

Although the three areas differed substantially in the number of cigarettes smoked per hour per 10 $m^2$, only ambient nicotine levels responded in a similar fashion. For example, the average smoking intensity (cigarettes per hour per 10 $m^2$) was 10.8 times greater in the smoking designated area than in the smoking-permitted floor. Similarly, the average ambient nicotine level was 15.6 times greater in the smoking designated than the smoking-permitted areas. Corresponding ratios for $CO_2$ and CO were only 1.3 and 1.7 respectively.

Smoking was not observed on the nonsmoking floor. The average ambient nicotine concentrations were below the limit of detection (i.e. less than 1.6 $\mu g/m^3$) for the one hour sampling periods used. CO and $CO_2$ levels were slightly lower on the nonsmoking floor than on the smoking-permitted floor.

Table 1 also lists the average person densities for each of the three sampling areas. The average person density was 30% higher on the smoking-permitted floor and 60% higher in designated smoking area than on the nonsmoking floor. Corresponding ratios calculated for CO and $CO_2$ show a similar pattern and range.

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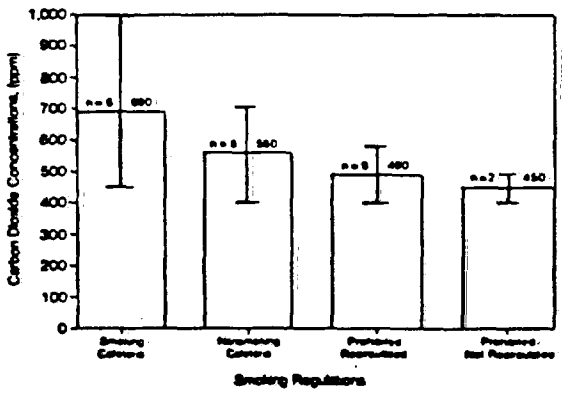
FIELD STUDY II

The data for the smoking and nonsmoking cafeteria sections of Buildings A and B were combined because there were only very small differences in the results for each building.

Figures 1 through 4 summarize the ambient $CO_2$, CO, RSP and nicotine concentrations, in four areas under different smoking and ventilation conditions: the smoking sections of the two cafeterias, the nonsmoking sections of the two cafeterias, the nonsmoking offices of Building A which received recirculated air from other areas of the building, and the separately ventilated nonsmoking offices of Building B which did not receive recirculated air from other areas of the building. In each figure, the height of the bars give the average concentration, n gives the total number of samples on which the average is based. The range of observed values is given by the vertical line.

Figure 1 shows that there was little difference between the $CO_2$ levels in the smoking prohibited offices of building A and building B, whether or not they received recirculated air. The smoking and nonsmoking sections of the cafeterias had slightly elevated, but not statistically significant, $CO_2$ concentrations when compared to the nonsmoking offices. These small increases in $CO_2$ levels could have been partly due to occupant density which was twice as high in the cafeteria sections than in the nonsmoking offices.

Figure 1
Comparison of Averages and Ranges of $CO_2$ Concentrations for Different Smoking Regulations




The distribution of mean ambient CO levels is similar to that for $CO_2$, as shown in Figure 2. Although mean CO levels were higher in both cafeteria sections than in the nonsmoking offices, none of the differences were statistically significant.

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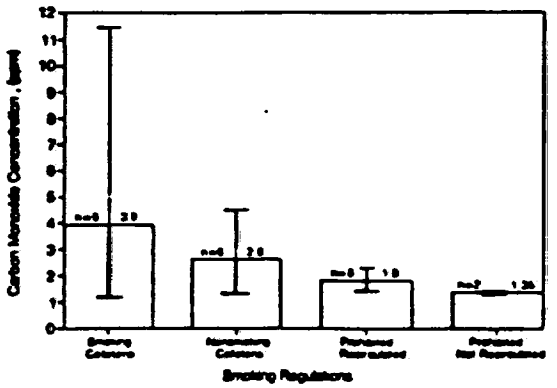
Figure 2

Comparison of Averages and Ranges of CO Concentrations for Different Smoking Regulations




There were no differences in RSP concentrations between the nonsmoking offices with and without recirculated air, as shown in Figure 3. However, the mean RSP level in the smoking section of the cafeterias was 2.6 times that in the nonsmoking offices while the mean level in the nonsmoking section of the cafeterias was about 1.7 times that found in nonsmoking offices.

Figure 3

Comparisons of Averages and Ranges of RSP Concentrations for Different Smoking Regulations

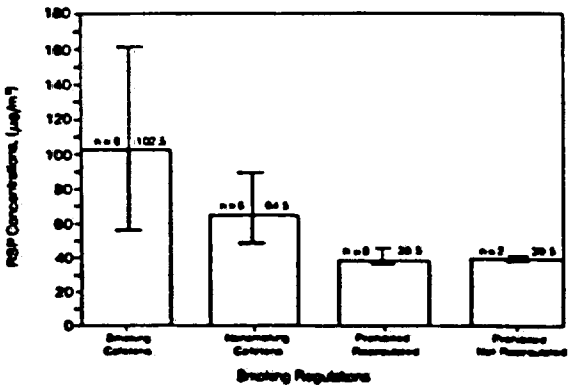



Figure 4 gives average ambient nicotine concentrations. In some instances the nicotine levels in the cafeteria sections were below the detection limit of 1.6 $\mu g/m^3$. The mean nicotine concentration measured in the smoking sections of the cafeterias was more than twice that in the nonsmoking sections of the cafeterias and at least 15 times that in the nonsmoking offices which received recirculated air.



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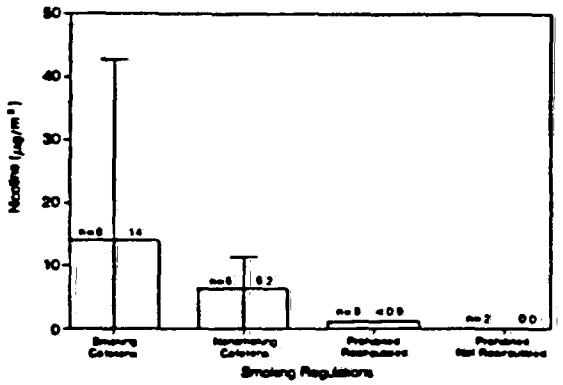
Figure 4

Comparison of Averages and Ranges of Nicotine Concentrations for Different Smoking Regulations




The lower detection limit for nicotine is dependent upon the amount of air that is sampled. For the method used in this study, the lower detection limit was 0.8 $\mu g/m^3$ for a two-hour sample, 0.4 $\mu g/m^3$ for a four-hour sample, and 0.2 $\mu g/m^3$ for an eight-hour sample. Two-, four-, and eight-hour samples were taken in nonsmoking offices in Building A which received recirculated air. None of four two-hour samples were above the detection limit; one of three four-hour samples was above the detection limit and gave a determination of 1.0 $\mu g/m^3$; the single eight-hour sample was also above the detection limit and yielded a determination of 0.8 $\mu g/m^3$. These results indicate that the ambient nicotine concentration in these nonsmoking offices was not larger than the maximum positive result of 1.0 $\mu g/m^3$.

Two two-hour nicotine samples were taken in nonsmoking offices in building B which did not receive recirculated air. Nicotine was below the detection limit of 0.8 $\mu g/m^3$ in both samples.

## DISCUSSION

The FIELD STUDY I results suggest that ETS contributes little to ambient $CO_2$ levels. The differences in $CO_2$ concentrations between the smoking-permitted, prohibited and designated smoking areas were small compared with the observed differences in smoking intensity (cigarettes smoked per hour per 10 $m^2$) and ambient nicotine levels. However, the sample sites with higher $CO_2$ levels in FIELD STUDIES I and II also had higher person densities. This suggests that people were the primary source of $CO_2$.

Ambient CO concentrations increased with smoking (Table 1 and Figure 2) but did not closely follow smoking intensity or nicotine concentrations. Other indoor and outdoor sources of CO must also contribute to CO levels, as indicated by the background level of 1.35 ppm in the nonsmoking office

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without air recirculation. Part of the higher CO level in the cafeteria could also be due to cooking activities.

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Not surprisingly, of the four substances measured, nicotine shows the strongest association with smoking. There are few if any significant sources of nicotine in the non-industrial indoor environment other than smoking and it follows that nicotine is an accurate marker of ETS exposure. Improvements in nicotine measurement technology could result in the widespread use of nicotine as an indicator of ETS exposure (4).

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Nicotine levels were at or below 1 $\mu g/m^3$ in nonsmoking offices which receive recirculated air from smoking designated areas. This level of exposure is very low. For example, breathing air which contains 1.0 $\mu g/m^3$ of nicotine for one hour at an average respiration rate for office activity of 0.48 $m^3/hr$ (5) is approximately equal to 1/1,900th of the 900 $\mu g$ of nicotine inhaled by a smoker from the mainstream smoke of one cigarette (6).

There are few published studies of nicotine levels in nonsmoking offices. The nicotine levels observed by Bayer and Black (7) are, unfortunately, not comparable with the results given here because their results are given in $\mu g/m^3/min$. Nevertheless, they did not detect nicotine in two of three nonsmoking offices.

Other office studies have also found low RSP levels in nonsmoking areas close to smoking designated areas that are not separately ventilated (8). These findings are reinforced by Health and Welfare Canada (1) who measured RSP, CO and $CO_2$ concentrations before and after the implementation of a no smoking policy in offices that received recirculated air from a designated smoking area. After the no smoking policy was implemented, mean RSP levels decreased by 8 $\mu g/m^3$, from 26 $\mu g/m^3$ to 18 $\mu g/m^3$ (HWC, 1985a).

Another Health and Welfare Canada study (9) measured RSP and $CO_2$ on three floors of an office building before and after the implementation of a no smoking policy. This study differed from the previous HWC study in that smoking was restricted to a separately ventilated area. Table 2 gives mean RSP concentrations for the three floors before and after the implementation of the no smoking policy. The average RSP concentration on the three floors decreased from 28.1 $\mu g/m^3$ to 21.1 $\mu g/m^3$, for a net reduction of 7 $\mu g/m^3$.

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TABLE 2

Comparison of RSP Mean Concentrations on Office Floors Before and After a No Smoking Policy was Implemented (Extracted from Health and Welfare Canada (9))

| | RSP ($\mu g/m^3$) | $CO_2$ (ppm) |
|---|---|---|
| Before No Smoking Policy | | |
| Floor A | 29.3 | 663 |
| Floor B | 30.0 | 614 |
| Floor C | 25.0 | 606 |
| Overall Average | 28.1 | 627 |
| After No Smoking Policy | | |
| Floor A | 22.7 | 591 |
| Floor B | 19.8 | 551 |
| Floor C | 20.8 | 503 |
| Overall Average | 21.2 | 551 |

The results of the FIELD STUDY II and the Health and Welfare Canada study (1) indicate that recirculated air from designated smoking areas contributes less than 8 $\mu g/m^3$ of RSP to nonsmoking offices which receive such air. Another Health and Welfare Canada study (9) further suggests that ad lib smoking in offices under normal ventilation and occupancy conditions contributes about 7 $\mu g/m^3$ of RSP. These findings are substantially different from the widely quoted estimate by Repace and Lowrey (10) that smoking increases RSP levels in offices by 170 to 200 $\mu g/m^3$. This estimate, also accepted by Health and Welfare Canada, is based on modelling exposures. However, Repace and Lowrey's estimate is up to 21 to 29 times greater than the observed effect of smoking on RSP levels in the two HWC studies and, in fact, is 5.3 to 6.3 times larger than the average RSP concentration of 31.8 $\mu g/m^3$ from all sources found in smoking designated areas in the Health and Welfare Canada study (9). These results further verify the results of a comparison of air pollutant measurements in a large number of buildings with and without smoking regulations (11). The study found only small differences in airborne particles (combined total and respirable particles).

While smoking regulations are here to stay and will affect most offices under federal, provincial or municipal control in Canada, the haste to regulate smoking may have been based on the unrealistic modelled estimates of Repace and Lowrey or on "worst case" measurements in poorly ventilated workplaces, instead of on actual measurements of RSP levels in typical offices.

The provision of a designated smoking area appears to effectively reduce ETS constituent levels in nonsmoking offices, even if the designated smoking area is not separately ventilated. However, we should caution that an exclusive reliance on regulating smoking while ignoring other sources of indoor pollution in the non-industrial work environment may accomplish little in meeting indoor air quality problems, especially in so-called "Sick Buildings" (11).

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REFERENCES

1. Health and Welfare Canada, Investigation on the Impact of a New Smoking Policy on Office Air Quality National Library of Canada Hull, Quebec. Occupational Health Unit Medical Services Branch (1985).

2. NIOSH, Manual of Analytical Methods, 2nd ed. US National Institute of Occupational Safety and Health, Cincinnati, Ohio (1977).

3. T.D. Sterling and B. Mueller, Concentrations of nicotine, RSP, CO, $CO_2$ in nonsmoking offices ventilated by air recirculated from smoking designated areas. Am Ind Hyg Assoc J (in press).

4. Health and Welfare Canada, Involuntary Exposure to Tobacco Smoke, Report of the Advisory Committee, Environmental Health Directorate, Ottawa (1987).

5. ASHRAE, ASHRAE Standard 62-1981R, Ventilation for acceptable indoor air quality. American Society of Heating, Refrigerating and Air-Conditioning Engineers, Atlanta, Georgia (1986).

6. M. Muramatsu, S. Umemura, T. Okada and H. Tomita, Environ Res. 35, 218-227 (1984)

7. C.W. Bayer and M.S. Black, Passive smoking: Survey analysis of office smoking areas vs. environmental chamber studies. Atlanta, Georgia (Proceedings, ASHRAE IAQ'86, pp. 281-291) (1986).

8. H.K. Lee, Investigation of the impact of a new smoking policy on office air quality in the National Library of Canada in Hull, Quebec. Ottawa, Proceedings of the 78th Annual Meeting of the Air Pollution Control Association (1985).

9. Health and Welfare Canada, Investigation on the Impact of a New Smoking Policy on Office Air Quality Office of the Auditor General Ottawa, Ontario. Occupational Health Unit Medical Services Branch (1985).

10. J.L. Repace and A.H. Lowrey, Science 208, 464-472 (1980)

11. T.D. Sterling, C.W. Collett and E.M. Sterling, J Occup Med 29, 57-62 (1987)

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# Concentrations of Nicotine, RSP, CO and $CO_2$ in Nonsmoking Areas of Offices Ventilated by Air Recirculated from Smoking Designated Areas

T.D. STERLING[A] and B. MUELLER[B]

[A]Faculty of Applied Sciences, Simon Fraser University, Burnaby, B.C. V5A 1S6 Canada;
[B]Theodor D. Sterling Limited, #70 - 1507 West 12th Avenue, Vancouver, B.C. V6J 2E2 Canada

**The exposure of nonsmokers to environmental tobacco smoke (ETS) when smoking is relegated to designated areas that are not separately ventilated is of considerable interest. Concentrations of nicotine, respirable suspended particles (RSP), carbon monoxide (CO), and carbon dioxide ($CO_2$) were measured in offices under different conditions of smoking regulation: smoking prohibited; smoking prohibited areas receiving recirculated air from designated smoking areas; smoking and nonsmoking sections of these designated smoking areas. Nicotine was collected by pumping air for periods of 1–8 hr at 1 L/min through sampling tubes containing a styrene divinylbenzene copolymer. RSPs (5 μm cut-off) were measured using an optical side scattering instrument. CO was measured by a direct reading electrochemical analyzer and $CO_2$ by colorimetric detector tubes. Detection of nicotine in nonsmoking office areas that received recirculated air from smoking designated areas required sampling times of 4 hr or more. Nicotine levels in such offices were approximately 1.0 μg/m³. RSP, CO and $CO_2$ concentrations were approximately the same in these offices as compared to nonsmoking offices not exposed to recirculated air from smoking areas. Providing a designated but not separately ventilated smoking area appears to be effective in eliminating most components of ETS from nonsmoking office work areas.**

## Introduction

A number of municipalities (San Francisco and Vancouver being leading examples) have passed bylaws to regulate smoking in public buildings. In principle these bylaws apply to public buildings and places of employment and establish a norm of no smoking except in smoking areas designated by the employer or proprietor. The Canadian and American Federal Governments are preparing to develop approaches to regulate smoking in workplaces under federal jurisdiction. Provincial and state governments are making similar preparations.

Four options are available to regulate office smoking:

1. Prohibiting smoking outright;
2. restricting smoking to designated areas that are ventilated separately;
3. restricting smoking to designated areas that are not ventilated separately; and
4. providing some framework by which an adjustment between smoking and nonsmoking workers may be achieved without directly regulating the placement of smokers.

The third option, that of providing a designated but not separately ventilated smoking area, appears to be the most frequently adopted procedure. A certain proportion of a building's population will demand a location where they may be allowed to smoke (for example, employees on their coffee and lunch breaks, members of the public waiting for services, or persons who are residents of the building—such as in prisons or hospitals.) Governments and the private sector own, operate and rent a wide variety of different buildings, however, most of these buildings do not offer separate ventilation for different locations. To provide separate ventilation would not only be costly in many instances but very often physically impossible. Thus, the least disruptive and costly solution for many buildings appears to be the setting aside of designated but not separately ventilated smoking areas.

A question of considerable interest is the extent to which designated but not separately ventilated smoking areas are effective in decreasing exposure to environmental tobacco smoke (ETS) in nonsmoking areas. This project was designed to provide some information on that question.

The authors report here the outcome of a series of measurements of nicotine, respirable suspended particles (RSP), carbon monoxide (CO) and carbon dioxide ($CO_2$) obtained in the following locations:

1) two cafeterias, each having smoking and nonsmoking areas;
2) four nonsmoking floors which received air recirculated from a ventilation system common to one of the cafeterias; and
3) two nonsmoking offices with independent ventilation systems which, therefore, did not receive air recirculated from designated smoking areas.

## Methods

Air sampling for nicotine, RSP, CO and $CO_2$ and an observation of the number of office occupants present and cigarettes smoked was undertaken in two adjacent buildings

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(Vancouver City Hall and City Hall Annex): Building A, which is a sealed, mechanically ventilated building, and Building B, which has opening windows and mechanical ventilation only in selected areas.

### Building Description

Building A is a 4-story sealed office building with 2 levels of underground parking. Each of the 4 floors contains approximately 1390 $m^2$ (15 000 $ft^2$) of office space. Fresh air from an intake at ground level is supplied to an air-handling unit in the basement mechanical room. This fresh air is filtered, conditioned and then supplied unmixed to air induction units located at exterior walls. Air is returned, via ceiling return grates, to a second air-handling unit in the basement, which exhausts a portion of the return air, adds makeup air (minimum of 20%), and filters, conditions and returns the air to the occupied space via ceiling diffusers. As a result, indoor air from different parts of the building and different floors is mixed. Smoking is prohibited in all work areas and public areas of the building and is permitted only in the smoking section of the fourth floor cafeteria which is not separately ventilated.

Building B is a 12-story, unsealed building with opening windows and, originally, no mechanical ventilation system. Most areas are passively ventilated by building leakage while separate ventilation systems have been incorporated in only a few areas. In the offices where measurements were taken, rooms with exterior walls have opening windows. Additional ventilation is supplied to the central zone of each of these offices by an air-handling unit which receives fresh air from an intake at ground level. The zone air-handling unit feeds conditioned air to a supply-air plenum (in the ceiling space) where individual fan-coil units temper the air again and deliver it to the occupied space below. Air which has not been exhausted through windows or doors is returned to the ceiling plenum and again tempered by the fan-coil units. These systems, therefore, have no ducting common to other areas of the building. Smoking is prohibited in all work areas and public areas in the building except the smoking section of the cafeteria (which is located in the basement). Heated/cooled air is supplied separately to the cafeteria and exhausted through windows.

### Sampling and Occupant Observation

Three samples were taken in each of the smoking and nonsmoking sections of the cafeterias of Buildings A and B; two samples on each of the four floors in nonsmoking offices of Building A; two samples in the nonsmoking offices of Building B; and two samples of RSP outdoors.

Samples for nicotine were obtained using a portable air sampling pump housed inside a briefcase. Because of the effect of air sampling on occupant behavior,[1] the sampling apparatus was designed to collect samples in an unobtrusive manner. Nicotine samples were collected by pumping air at 1 L/min through sorbent tubes containing XAD-4 resin, a styrene divinylbenzene copolymer. The sorbent tubes contained 80 mg of resin in the front (primary) section and 40 mg in the rear (secondary) section. Samples were collected for 1 hr each in the cafeteria locations and for periods of 2, 4 or 8 hr at other sampling sites. Respirable suspended particles (5 $\mu$m cutoff) were determined using a P-5H digital dust indicator (Sibata Scientific Technology, Tokyo, Japan) which measures light side-scattered by suspended particles. The unit was calibrated at the factory to monodispersed stearic acid particles with a mean diameter of 0.3 $\mu$m. The unit usually measured respirable particles for the entire sampling period, depending on battery charge. Approximately midway into the 1- or 2-hr air sampling period, CO and $CO_2$ concentrations were measured at the sampling locations. (CO and $CO_2$ were measured more often during 4- and 8-hr sampling periods.) CO was measured using a direct-reading electrochemical analyzer (Nova 310 L, Nova Analytical Systems, Inc., Hamilton, Ontario) housed in a flight case. $CO_2$ was measured using colorimetric detector tubes (Gastec, Gastec Corporation, Yokohama, Japan) and a manual sampling pump.

During a sampling period, the number of occupants in a predefined observation area and the number of cigarettes smoked in that area were observed and recorded. The observation areas were defined by visual configurations and reliability of surveillance of the observable office area. For purposes of comparison, number of persons and cigarettes smoked were calculated per 10 $m^2$ where applicable. At the completion of sampling, the sorbent tubes were refrigerated until analysis.

### Analysis

In the chemical analysis of nicotine, resin beads in the sorbent tubes were transferred to gas chromatograph autosampler vials to which were added 50 $\mu$L of quinoline (100 mg/L) to serve as an internal standard and 1 mL of ethyl acetate as an extraction solvent. Triethylamine (0.01% by volume) was added to the extraction solvent to prevent adsorptive losses of nicotine onto the glass autosampler vials. Samples and spiked standards then were placed on an automatic shaking device and shaken for 30 min. A Hewlett-Packard Model 5880A or Model 5830A gas chromatograph equipped with a nitrogen-phosphorus detector was employed in conjunction with an autosampler and a GC terminal to determine peak areas of the nicotine and compare them with the areas obtained from nicotine standards. The assayed nicotine was corrected for the desorption efficiency (usually 94%) of the particular lot of XAD-4 resin used in sampling. Final nicotine results were divided by the volume of air sampled to yield results in $\mu g/m^3$. The rear (backup) sections of sorbent tubes were analyzed separately and, except for one case, always yielded nicotine determinations less than the limit of detection, thus indicating no breakthrough of nicotine past the primary section. [The authors' procedure, by and large, is based on the National Institute of Occupational Safety and Health (NIOSH) method.[2]]

Respirable suspended particles were estimated by converting the digital counts of particles per sampling time to an average count per minute. A background count of 5 counts per min was subtracted from the average to yield RSP values in $\mu g/m^3$.

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## Results

Table I summarizes measurements for RSP, CO, $CO_2$, nicotine; average number of persons per 10 $m^2$; and average number of cigarettes smoked per hour per 10 $m^2$ (where applicable). Because of the large variability and suspected skew of measures, means, medians and ranges are given. Measurements in the cafeteria smoking areas each are based on 6 samples as are measurements in the cafeteria nonsmoking areas. Because there were no perceptible differences between cafeterias in Buildings A and B, both for smoking and nonsmoking areas, their data have been merged. Measurements in nonsmoking office areas in Building A are based on 8 samples, and measurements in nonsmoking areas in Building B are based on 2 samples.

There were significantly more persons per unit area in the cafeterias than in the nonsmoking offices. The numbers of individuals per 10 $m^2$ in smoking and nonsmoking areas of cafeterias, however, were approximately the same. As might be expected, both CO and $CO_2$ levels were higher in the smoking than nonsmoking areas of the cafeterias. This also was true for RSPs. Nicotine levels averaged 14.0 $\mu g/m^3$ in the smoking area and 6.2 $\mu g/m^3$ in the nonsmoking area of the cafeterias. The drop in RSPs and nicotine from smoking to nonsmoking areas of the cafeterias is quite steep and attests to the rapid dilution of ETS.

Contributions to RSP, CO and $CO_2$ that are caused by smoking in the designated smoking area are diluted further in the recirculated air. This dilution can be seen from a comparison of measurements in the office areas of Building A with Building B. Concentrations of RSP, CO and $CO_2$ in Building A's nonsmoking areas, which received recirculated air from the smoking area, are approximately the same as those measurements taken in Building B, which did not receive any such recirculated air (also see Table II). Of special interest are measurements of nicotine. It is important to keep in mind that the detection of nicotine in air, in the dilute quantities in which it may be present, requires a lengthy sampling procedure. As the concentration of nicotine in air decreases, larger air samples must be obtained to detect that concentration. For the method used here, a 2-hr sample at 1 L/min would detect nicotine concentrations greater than 0.8 $\mu g/m^3$. Of 4 samples taken for 2 hr each, not a single sample detected a concentration above 0.8 $\mu g/m^3$. For a 4-hr sample at 1 L/min, the lower level of detection is 0.4 $\mu g/m^3$. At that level, 1 positive detection at a concentration of 1.0 $\mu g/m^3$ was made in 1 out of 3 samples. For the 1 sample taken for 8 hr, the lower level of detection was 0.2 $\mu g/m^3$. That sample measured a concentration of 0.8 $\mu g/m^3$ (findings summarized in Table II).

## Discussion

Studies of office air quality have demonstrated that significant reductions in ETS related RSP may be achieved in nonsmoking areas when smoking is limited to designated areas that are not ventilated separately.[1] The extent of involuntary exposure to ETS, however, best may be established quantitatively when nicotine is used as the marker.* It has been suggested[3] that advances in measurement technology may provide grounds for reliance on nicotine as a general indicator of ETS. Other components of ETS may be less useful for developing an ETS exposure index. ETS components are complex and variable and also include many constituents similar to those emitted from other sources.[4]

*The observation that nicotine in sidestream smoke is mainly in the vapor phase while in mainstream smoke it is more in the particulate (deposit) phase poses no obstacle to the use of nicotine as an index of ETS infiltration because building occupants are not exposed to mainstream smoke unless they actively do smoke. The nicotine concentration obtained from sampling the air is a representative sample of ambient ETS inhaled by nonsmokers.

**TABLE I**
**Comparison of ETS Related Air Quality Parameters in Nonsmoking Work Areas and Designated Smoking Areas**

| | | RSP[A] ($\mu g/m^3$) | CO (ppm) | $CO_2$ (ppm) | Nicotine ($\mu g/m^3$) | Persons /10 $m^2$ | Cigarettes /hr/10 $m^2$ |
|---|---|---|---|---|---|---|---|
| Smoking areas of Cafeterias A & B combined | Mean | 70 | 3.9 | 690 | 14 | 1.8 | 1.2 |
| | Range | 23–129 | 1.1–11.4 | 450–1000 | <1.6–43.7 | 0.79–3.42 | 0.53–1.67 |
| | Median | 74 | 2.5 | 650 | 11 | 1.6 | 1.2 |
| Nonsmoking areas of Cafeterias A & B combined | Mean | 32 | 2.6 | 560 | 6.2 | 1.7 | |
| | Range | 15–57 | 1.2–4.5 | 400–700 | <1.6–10.9 | 0.76–2.5 | NA[B] |
| | Median | 26 | 2.4 | 580 | 7.9 | 1.7 | |
| Nonsmoking office area, Building A | Mean | 6 | 1.8 | 490 | [C] | 0.73 | |
| | Range | 4–11 | 1.3–2.3 | 400–580 | | 0.28–1.9 | NA |
| | Median | 6 | 1.7 | 500 | | 0.46 | |
| Nonsmoking office area, Building B | Mean | 7 | 1.35 | 450 | [C] | 0.9 | |
| | Range | 6–8 | 1.3–1.4 | 400–500 | | 0.53–1.28 | NA |
| | Median | 7 | 1.35 | 450 | | 0.9 | |

[A]Mean outdoor RSPs were 10 $\mu g/m^3$.

[B]NA = not applicable

[C]See Table II

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**TABLE II**
**Nicotine, RSP, CO and $CO_2$ Concentrations in Eight Locations in a No Smoking Office Area that Receives Recirculated Air from a Smoking Designated Area and in Two Locations without Such Recirculation[A]**

| | Location | Sample Time (hr) | Nicotine ($\mu g/m^3$) | RSP ($\mu g/m^3$) | CO (ppm) | $CO_2$ (ppm) | Persons /10 $m^2$ |
|---|---|---|---|---|---|---|---|
| Recirculated air | 1 | 2 | <0.8 | 6 | 1.7 | 580 | 0.50 |
| | 2 | 2 | <0.8 | 5 | 2.3 | 500 | 0.42 |
| | 3 | 2 | <0.8 | 5 | 1.3 | 400 | 1.90 |
| | 4 | 2 | <0.8 | 4 | 2.0 | 500 | 0.38 |
| | 5 | 4 | <0.4 | 11 | 2.2 | 550 | 0.39 |
| | 6 | 4 | <0.4 | 5 | 1.7 | 450 | 0.28 |
| | 7 | 4 | 1.0 | 6 | 1.7 | 500 | 1.02 |
| | 8 | 8 | 0.8 | 6 | 1.6 | 450 | 0.96 |
| No recirculated air | 9 | [B] | [B] | 8 | 1.4 | 400 | 0.53 |
| | 10 | [B] | [B] | 6 | 1.3 | 500 | 1.28 |

[A]Sampling time for nicotine ranged from 2 to 8 hr.

[B]Nicotine was not measured because these offices could not receive ETS from any source.

Air sampled for 2 hr at 1 L/min (using the NIOSH protocol) reliably measures nicotine levels that are larger than 0.8 $\mu g/m^3$. Levels of nicotine appear to be at or below that concentration in offices in which smoking is prohibited but which receive air recirculated from smoking designated areas. To give meaning to such trace values, the exposure of an office worker to nicotine at 1 $\mu g/m^3$ for 1 hr can be calculated roughly. Given a breathing rate of 0.48 $m^3$/hr for the level of activity required during normal office work,[5,6] an office worker would breath air containing 0.48 $\mu g$ of nicotine in 1 hr. This quantity is approximately equivalent to 1/1800 of the nicotine inhaled by actively smoking 1 cigarette (900 $\mu g$/cigarette[6]). Until relatively recently, calculations of a smoker's exposure to cigarette smoke was limited to amounts of materials in the mainstream smoke. Insofar as smokers are spatially close to their cigarette and often inhale relatively undiluted sidestream smoke, existing estimates of smokers' exposure to any component of ETS must be lower than their actual magnitudes. Thus, the nonsmoker probably inhales less than 1/1800 of the nicotine inhaled by a smoker when actively smoking one cigarette, unless this nonsmoker should be standing in very close proximity to a burning cigarette.

Based on these findings, it is the authors' belief that the provision of a designated smoking area appears to be effective in eliminating most traces of ETS from the rest of the office space, even if the designated smoking area is not separately ventilated. An exclusive reliance on regulating smoking while ignoring all other problems besides smoking which may influence the quality of air in the nonindustrial work environment may accomplish little in addressing indoor air quality problems, however, especially in so-called "sick buildings."[7]

If a designated area is made available for smoking in offices where otherwise smoking is not permitted, the designated space should be sufficiently large to prevent overcrowding.

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**References**

1. **Lee, H.K., T.A. McKenna, L.N. Renton and J. Kirkbride:** Investigation of the Impact of a New Smoking Policy on Office Air Quality in the National Library of Canada in Hull, Quebec. In *Proceedings of the 78th Annual Meeting of the Air Pollution Control Association, Ottawa,* 1985.
2. **National Institute for Occupational Safety and Health:** *Manual of Analytical Methods.* 2nd Ed. Cincinnati, Ohio: United States Department of Health, Education and Welfare/National Institute of Occupational Safety and Health, 1977. pp. S293-1-S293-9.
3. **National Health and Welfare, Canada:** Involuntary Exposure to Tobacco Smoke. Report of the Advisory Committee. Ottawa, Canada: Environmental Health Directorate, 1987.
4. **Bayer, C.W. and M.S. Black:** Passive Smoking: Survey Analysis of Office Smoking Areas *vs.* Environmental Chamber Studies. In *IAQ '86: Managing Indoor Air for Health and Energy Conservation.* Atlanta, Ga.: American Society of Heating, Refrigerating and Air-Conditioning Engineers, 1986. pp. 281-291.
5. **American Society of Heating, Refrigerating and Air-Conditioning Engineers, Inc.:** *ASHRAE Handbook: 1985 Fundamentals.* Atlanta, Ga.: American Society of Heating, Refrigerating and Air-Conditioning Engineers, Inc., 1985. pp. 8.11-8.12.
6. **Muromatsu, M., S. Umemmura, T. Okada and H. Tomita:** Estimation of Personal Exposure to Tobacco Smoke with a Newly Developed Nicotine Personal Monitor. *Environ. Res.* 35:218-227 (1984).
7. **Sterling, T.D., C.W. Collett and E.M. Sterling:** Environmental Tobacco Smoke and Indoor Air Quality in Modern Office Work Environments. *J. Occup. Med.* 29:57-62 (1987).

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# EXPOSURE TO ENVIRONMENTAL TOBACCO SMOKE IN THE NON INDUSTRIAL WORKPLACE UNDER DIFFERENT CONDITIONS OF VENTILATION AND SMOKING REGULATION

TED STERLING

Faculty of Applied Science, School of Computer Science, Simon Fraser University, Burnaby, B.C., V5A 1S6

CHRIS COLLETT and JAMES ROSS

Theodor D. Sterling and Associates Ltd., 20 - 1507 West 12th Avenue, Vancouver, B.C., V6J 2E2

## INTRODUCTION

This report presents the results of three field studies examining levels of environmental tobacco smoke (ETS) in office buildings under conditions of normal occupancy, smoking and ventilation. Nicotine, Respirable Suspended Particles (RSP), Carbon Monoxide (CO) and Carbon Dioxide ($CO_2$) were measured as markers of ETS. One or more of these substances have frequently been measured in investigations of smoking and nonsmoking offices.(1,2) Of these four substances, only nicotine is uniquely related to the combustion of tobacco. RSP concentrations vary with different types of human activities found in office buildings. CO and $CO_2$ are byproducts of tobacco combustion which also have multiple other sources. A main source of CO in office buildings is the infiltration of automobile exhaust. The primary sources of $CO_2$ are the outdoor air and the exhaled breath of occupants.

The field studies were designed to evaluate two questions:

1. What is the contribution of smoking to indoor ETS concentrations (as measured by nicotine, RSP, $CO_2$ and CO)?
2. How does the restriction of smoking to specially designated, but not separately ventilated, areas effect the levels of ETS in nonsmoking office spaces?

## SAMPLING AND ANALYTICAL METHODS

### Nicotine

Indoor nicotine samples were collected using a air sampling pump housed inside a briefcase. Samples were collected by pumping air

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at 1 liter per minute through sorbent tubes containing XAD-4 resin, a styrene divinylbenzene copolymer. The sampling techniques collects gaseous phase nicotine, rather than particulate phase. Collected samples were analyzed with a gas chromatograph equipped with a nitrogen-phosphorous detector, using a procedure based on a standardized NIOSH analytical method.(3)

RSP, CO and CO2

Instantaneous measurements for RSP, CO and $CO_2$ were taken during the nicotine sampling periods. Measurements of each substance were taken at the start and completion of each nicotine sampling period, and at one hour intervals throughout the intervening time. The data were averaged to provide a representative value for each sampling period.

RSP concentrations were measured with a Sibata P-5H Digital Dust Indicator, which determines RSP concentrations as a function of the intensity of scattered light as respirable particles (< 3.5 μm) are drawn through a sensing chamber. The instrument had been calibrated to ETS particles through side by side comparison with gravimetric sampling.

CO concentrations were measured using a direct reading electrochemical analyzer (NOVA 310L), $CO_2$ was measured using extra low range colorimetric detector tubes.

Other Observations

During each field investigation, observations of occupant behavior were also made. In Studies One and Three, the number of occupants and the number of cigarettes smoked in each predefined observation area were recorded for the entire sampling period. In Study Two, the number of cigarettes being smoking at the time of the instantaneous measurements of RSP, CO and $CO_2$ were observed, and a mean value calculated.

RESULTS

Study 1

The first field study was undertaken in an 14-storey office building. Measurements of nicotine, CO and $CO_2$ were taken on the 7th and 11th floors of the building. (RSP measurements were also analyzed, but not recorded due to instrument failure.) Smoking was restricted to a 22.5 $m^2$ lounge on the 7th floor, without separate ventilation. No smoking was allowed elsewhere on the 7th floor.

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On the 11th floor, smoking was permitted without restriction. The layout and design of the two floors were similar with an open plan office area of 780 $m^2$ surrounding a central core, containing mechanical rooms and service shafts.

Each floor of the building was independently ventilated. Outside air was ducted from a rooftop intake to a mechanical room on each floor. An air handling unit in the mechanical room mixed outside air with return air from the office space, then filtered and heated or cooled the mixed air before suppling it to the office space. Supply air was ducted through the suspended ceiling to rectangular ceiling diffusers. The suspended ceiling was also used as the return air plenum. On the days of sampling, the system was introducing 15-20% outside air to the space. Therefore, a proportion of the air exhausted from the smoking lounge was recirculated into the nonsmoking office area.

Table 1 presents the mean and range of concentrations of nicotine, CO, $CO_2$, the average number of persons per 10 $m^2$, and the number of cigarettes smoked per hour per 10 $m^2$ in the three sampling locations.

TABLE 1

COMPARISON OF ETS RELATED AIR QUALITY PARAMETERS: FIELD STUDY 1

| | Nicotine ($ug/m^3$) | CO (ppm) | $CO_2$ (ppm) | Persons/10$m^2$ | Cig/hr/ 10$m^2$ |
|---|---|---|---|---|---|
| FLOOR 11 | | | | | |
| Smoking | 4.9 | 2.5 | 720 | 0.79 | 0.36 |
| Permitted | <1.6*-8.0 | 0.9-5.3 | 500-900 | 0.35-1.37 | 0-0.71 |
| FLOOR 7 | | | | | |
| Smoking | <1.6 | 2.1 | 680 | 0.61 | NA |
| Prohibited (recirc) | <1.6-3.0 | 0.6-3.5 | 500-900 | 0.37-0.94 | NA |
| FLOOR 7 | | | | | |
| Smoking | 75.0 | 4.3 | 970 | 0.94 | 4.00 |
| Lounge | 33.0-210 | 2.5-6.2 | 750-1100 | 0.22-2.11 | 1.78-5.78 |

* Level of detection for a 1 hour sample.

Although the two areas in which smoking occurred differed substantially in the number of cigarettes/hr/10 $m^2$, only nicotine concentrations responded in a similar fashion. The average smoking

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intensity (cigarettes/hr/10 $m^2$) was 10.8 times higher in the smoking designated area than in the smoking-permitted floor. Similarly, the mean nicotine level was 15.6 times higher in the designated smoking lounge, than compared to the smoking-permitted areas.

Smoking was not observed in the nonsmoking areas on floor 7. Nicotine concentrations were below the limit of detection for the one hour sampling periods (<1.6 $\mu g/m^3$) , with one exception. In one of the 10 sampling periods, a nicotine concentration of 3 $\mu g/m^3$ was measured.*

CO and $CO_2$ concentrations were highest in the smoking lounge, and similar in both the smoking prohibited and smoking permitted office areas.

Study 2

Field study two compared ETS levels in a cafeteria used as a smoking lounge and in an adjacent nonsmoking staff lounge served by the same ventilation system in a four storey building. The floor areas of the smoking lounge and the nonsmoking staff area were 65 $m^2$ and 46 $m^2$ respectively.

The ventilation system serving the two study areas was a Variable Air Volume (VAV) system in which outside air was supplied to an air handling unit located in a rooftop penthouse. Outside and return air were mixed by the air handling unit and supplied to the space. On the days of measurement, between 10 and 30% of the return air was being recirculated. Air was ducted from the air handling unit through VAV boxes to slot diffusers in the suspended ceilings of the two study areas. Return air from the space was passively exhausted into a common ceiling plenum through linear slots. A common return fan drew air from the plenum back to the air handling unit. By using the ceiling as the return air plenum, air exhausted from the smoking lounge may be recirculated to other locations served by the same air handling unit, such as the staff lounge.

*In view of the fact that nine of the ten samples were below the level of detection (1.6 $\mu g/m^3$), the single detected value may be questionable, possibly due to an unobserved smoker or analytical error.

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Table 2 shows the mean and ranges of ETS related contaminants in the smoking lounge and nonsmoking staffrooms.

TABLE 2

COMPARISON OF ETS RELATED AIR QUALITY PARAMETERS: FIELD STUDY 2

| | Nicotine ($ug/m^3$) | RSP ($ug/m^3$) | $CO_2$ (ppm) | # of Cigs Smoked* |
|---|---|---|---|---|
| Cafeteria/ Smoking Lounge | 5.8<br>3.8-7.2 | 60.4<br>27-82 | 500<br>400-600 | 7.1<br>2-15 |
| Nonsmoking Lounge (recirc) | 0.6<br><0.4**-0.8 | 20.8<br>17-26 | 403<br>350-450 | --<br>-- |
| Outdoors | --<br>-- | 15.0<br>13-16 | 358<br>300-400 | --<br>-- |

* The number of cigarettes being smoked at the time of instantaneous measurements was recorded and a mean calculated.
**Level of detection for a 4 hour sample.

Mean nicotine concentration in the smoking lounge was 5.8 $\mu g/m^3$ Nicotine was measured at concentrations just above the detection limit (< 0.4 $\mu g/m^3$) in 3 of the 4 samples taken in the nonsmoking staff lounge, suggesting a low level of recirculation.

RSP concentrations were 2.9 times higher in the smoking lounge than in the nonsmoking areas. $CO_2$ concentrations were 1.2 times higher.

Study 3

In the third study, ETS related parameters were measured in two adjacent buildings, which contained a mixture of open-area offices, private offices and public waiting/service areas.

Building A was a sealed, mechanically ventilated four storey office building with two levels of underground parking. The building was served by a central ventilation system. Outside air was drawn into an air handling unit located in a basement mechanical room, mixed with return air and supplied to each floor through ceiling diffusers. Return air was drawn from the ceiling plenum back to the basement air handling unit. The amount of air recirculated by the system varied from 0 to 80%, depending on

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outdoor temperatures. At the time of survey, approximately 30 to 50% of the return air was recirculated.

Smoking was prohibited in all areas of Building A, except for a smoking section in the fourth floor cafeteria, which was not separately ventilated from the rest of the building or physically separated from the nonsmoking section. Therefore, return air from the smoking area could be recirculated throughout the building.

Building B was a 12-storey unsealed office building without mechanical ventilation. Most areas were therefore passively ventilated by building leakage. The building was heated by a hot water radiant system. Consequently, there was no mixing of air from different floors or offices. Smoking was prohibited in all areas of the building except in the smoking section of a basement cafeteria. No air from the cafeteria could be recirculated elsewhere in the building.

Table 3 summarizes the measurement results. As there were no perceptible differences between the measured concentrations in cafeterias in Buildings A and B, either for smoking and nonsmoking areas, their data has been merged.

TABLE 3

COMPARISON OF ETS RELATED AIR QUALITY PARAMETERS: FIELD STUDY 3

| | Nicotine ($ug/m^3$) | RSP* ($ug/m^3$) | CO (ppm) | $CO_2$ (ppm) | Pers/ $10m^2$ | Cigs/ $hr/10m^2$ |
|---|---|---|---|---|---|---|
| Cafeteria Smoking Sec. (A & B comb) | 14.0<br><1.6-43.7 | 70<br>23-129 | 3.9<br>1.1-11.4 | 690<br>450-1000 | 1.8<br>.8-3.4 | 1.2<br>.5-1.7 |
| Cafeteria Nonsmok Sec. Areas (A & B comb) | 6.2<br><1.6-10.9 | 32<br>15-57 | 2.6<br>1.2-4.5 | 560<br>400-700 | 1.7<br>.8-2.5 | NA<br>NA |
| Nonsmoking Offices Bldg A (recirc) | <0.8<br><0.4-1.0 | 6<br>4-11 | 1.8<br>1.3-2.3 | 490<br>400-580 | 0.73<br>.3-1.9 | NA<br>NA |
| Nonsmoking Offices Bldg B (no recirc) | --<br>-- | 7<br>6-8 | 1.35<br>1.3-1.4 | 450<br>400-500 | 0.9<br>.5-1.3 | NA<br>NA |

* Mean outdoor RSP's were 10 ug/m3.

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There was a higher occupant density in the cafeterias than in the nonsmoking office areas. However, the number of individuals per 10 $m^2$ in the smoking and nonsmoking sections of the cafeterias were approximately the same. Measured levels of all four air quality parameters were higher in the smoking sections than in the nonsmoking sections of the cafeterias. The decrease in RSP and nicotine concentrations between the smoking and nonsmoking areas of the cafeterias illustrates a rapid dilution of ETS.

Eight nicotine samples were collected in the nonsmoking offices of Building A. Measurable levels of nicotine were found in 2 of the 8 samples, indicating a low level of ETS infiltration.

## DISCUSSION

The results from the three field studies suggest that ETS contributes little to indoor $CO_2$ levels. Differences in $CO_2$ concentrations between the smoking-permitted, prohibited and designated smoking areas were small when compared with the observed differences in smoking intensity and nicotine levels. The sample sites with highest $CO_2$ levels in the four studies were those areas with the higher person densities.

Indoor CO concentrations increased with smoking but did not closely follow smoking intensity or nicotine concentrations. Other indoor and outdoor sources must also contribute to CO levels in buildings, as indicated by the background levels measured in areas which do not receive air recirculated from smoking lounges.

RSP concentrations were substantially higher in the smoking than nonsmoking areas. There were few differences between RSP concentrations measured in nonsmoking areas that received ,or did not receive, air recirculated from the smoking lounge. This finding suggests that recirculation of ETS can not be detected by elevated RSP concentrations.

Of the four substances measured, nicotine showed the strongest association with smoking. There are few, if any, significant sources of nicotine in the non-industrial indoor environment other than smoking. The results of the field studies clearly indicate that nicotine is the most accurate marker of the presence of ETS.

Nicotine was detected in six of 21 samples taken in nonsmoking areas that received recirculated air. In those cases where nicotine was detected, concentrations were more than 10 times lower than in the smoking lounge. These measurements indicate that ETS

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components are substantially diluted as they are recirculated from smoking designated to smoking prohibited work areas. Of course what is "substantial" dilution is to some extent subjective. To give meaning to such trace values, it has been demonstrated that the exposure of an office worker to recirculated nicotine is at, or below, 1 $\mu g/m^3$ for one hour. Giving a breathing rate of 0.48 $m^3/hr$ for the level of activity required for normal office work (4), an office worker would breath air containing 0.48 µg of nicotine in one hour. This quantity is approximately equivalent to 1/1800 of nicotine inhaled by actively smoking one cigarette (900 µg/cigarette. (5)

Overall, based on the results of the field studies, it appears that the provision of designated smoking areas in the four buildings has acted to effectively reduce ETS constituent levels in nonsmoking offices, even when the designated smoking areas are not separately ventilated.

REFERENCES

1. Lee HK (1985) Investigation of the impact of a new smoking policy on office air quality in the National Library of Canada. Proceedings: 78th Annual Meeting of the Air Pollution Control Association, Ottawa, Ontario

2. Miesner EA, Rudnich SN, Hu FC, Spengler JD, Preller L, Ozkaynak H, Nelson W (1988) Aerosol and ETS sampling in public facilities and offices. Proceedings: 81st Annual Meeting of the Air Pollution Control Association, Dallas, Texas

3. NIOSH (1977) Manual of Analytical Methods (2nd ed), U.S. National Institute of Occupational Safety and Health, Cincinnati, Ohio

4. ASHRAE (1988) ASHRAE Standard 62-1981R: Ventilation for Acceptable Indoor Air Quality (Proposed Revision). American Society of Heating, Refrigerating and Air-Conditioning Engineers, Atlanta, Georgia

5. Muramatsu M, Umemura S, Okada T, Tomita H (1984) Environmental Research 35:218-227

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# Air quality, ventilation, temperature and humidity in aircraft

*These four parameters were measured on 45 flights and compared with existing comfort regulations*

**By Anne O'Donnell, Giovanna Donnini and Van Hiep Nguyen**

*Affiliate Member ASHRAE*

The increasing number of air travelers in recent years has increased the concern of air quality in aircraft. Hence, it is vitally important that the health and comfort of passengers are ensured.

The health and comfort of air travelers depends on the complex interplay of several factors. Some important factors are: the adequacy of the ventilation system to allow fresh air in the occupied zone; the concentration of contaminants; the temperature; and relative humidity.

The purpose of our study was to measure these parameters, compare them with existing regulations and comfort recommendations, and then evaluate the health and comfort in passenger airplanes.

### Testing environment & strategy

The tests were conducted on 45 flights from seven identical aircraft of the same airline. The average flight duration was between 30 minutes and one hour; all were non-smoking flights.

The aircraft had a seating capacity of 101 passengers, with five-member crews. One or both of the aircraft conditioning units (hot air packs) were functioning on all flights, as selected by the flight crew.

About the authors

**Anne O'Donnell** is an industrial hygienist with Canadian Marconi Company, Montreal, Quebec. She received a B.S. from McGill University-Montreal and an M.S. in occupational hygiene from the University of Newcastle upon Tyne, England.

**Giovanna Donnini** is a student in the master program of applied science in building engineering at Concordia University-Montreal. She received a bachelors of engineering from Concordia University.

**Van Hiep Nguyen** is a researcher with the School of Occupational Health, McGill University. He received a bachelors of engineering from the University of Sherbrooke and a masters degree in occupational health from the University of Quebec.

Measurements were taken at two locations in the aircrafts' economy section: on the right side (when facing forward), in front of the first row; and on the left side, in front of the last row. The sampling strategy and methodology employed are summarized in *Table 1.*

The sampling and analysis of the chemical and gas contaminants were performed according to the National Institute of Occupational Safety and Health Guidelines (NIOSH 1977) and IRSST methods. The only exception was formaldehyde, which was sampled and analyzed using McGill University methods (an American Industrial Hygiene Association approved lab).

The quantity of fresh air was measured using the sulfur hexafluoride ($SF_6$) tracer gas method. A quantity of 1 ppm of $SF_6$ was injected along the aisle of the cabin. Air samples were taken 15 minutes following the $SF_6$ injection, every 3 to 5 minutes up to one hour. Air sampling started approximately 10 minutes after take-off. The $SF_6$ concentrations were

**Table 1. Summary of Sampling Methodology and Strategy**

| Contaminant/ Parameters | Measurement Techniques | Detection Limits | Sampling Frequency |
|---|---|---|---|
| Carbon monoxide | Direct reading electrochemical cell monitor | 1 ppm | Every 5 min. during each flight |
| Carbon dioxide | Direct reading infrared monitor | 10 ppm | Every 3-5 min. during each flight |
| Nitrogen dioxide | Direct reading electrochemical cell monitor | 0.05 ppm | Twice per flight |
| Formaldehyde | Passive sampler analyzed by gas chromatography | 17 ppm | Integrated sample taken in the test aircrafts' day schedule (6 to 8 hours) |
| Volatile organic compounds | Activated charcoal tube analyzed by gas chromatography | 25 μg | Tube sampled at 0.5 liters per minute over the duration of the test aircrafts' day schedule (6 to 8 hours) |
| Total particulates | Polyvinyl chloride filter 37 mm diameter; 0.8 μm pore size gravimetric analysis | 25 μg | Filter sampled at 1.5 liters per minute over the duration of the test aircrafts' day schedule (6 to 8 hours) |
| Temperature | Passive logger (resistor-thermistor series) | 1°C | Data recorded every 2 min. over the duration of each flight |
| Relative humidity | Passive logger (resistor-thermistor series) | 1% | Data recorded every 2 min. over the duration of each flight |
| Outdoor air supply | 60 cc syringe analyzed by gas chromatography | 5 ppb | Gas decay sampled with vacutainer tubes monitored up to every 5 min. for up to one hour, successfully on 33 flights |

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measured by gas chromatography with electron capture detection.

The calculation of fresh air change is related to the logarithmic degradation of the tracer gas concentration with respect to time. The slope of the curve is the rate of fresh air-change.

The calculation is performed using the following equations:

$$\ln C/C_1 = -(Q/V) \cdot T$$
$$\ln C/C_1 = -I \cdot T$$

where, $C_1$ = the initial tracer gas concentration (uniformly mixed) (ppm)

- C = tracer gas concentration after time T (ppm)
- Q = rate of fresh-air change of the aircraft ($m^3$/hour)
- V = the cabin volume ($m^3$)
- T = the time in hours
- I = the rate of fresh air-change per hour

Air temperature (which is controlled by the flight crew) and humidity (which originates from the passengers) were measured with four loggers. The two loggers were placed in the seating area behind each first class section and at the rear of each economy section.

Air temperature was measured at three levels: the floor; above the ankle; and at head height. This was approximately 0 in. (0 cm), 4 in. (10 cm), and 4.5 ft (140 cm) above the floor-level, respectively. Measurements were recorded every four minutes.

## Regulations & standards

The U.S. Federal Aviation Administration (FAA) applies limits for three contaminants: carbon monoxide, carbon dioxide and ozone. For ventilation, the FAA regulations state that the crew compartment should receive 10 cfm/person to enable crew members to perform their duties without undue discomfort or fatigue and that the passenger compartment "must be suitably ventilated." There are no specific limits for outdoor supply air.

The Aviation and Occupational Safety and Health Regulations of the Canadian Labour Code (CLC) use the American Conference of Governmental Industrial Hygienists (ACGIH) 1986-87 threshold limit values (TLV) in determining contaminant control limits. The concentration limits placed by the regulations are for health risk concerns (see *Table 2* for a summary of the regulations).

ASHRAE *Standard 62-1989* provides standards for outdoor supply air in transportation vehicles, and carbon dioxide levels and recommendations for comfort. Other standards (see *Tables 2* and *3*) are also used as comfort guidelines. All are usually used in evaluating comfort in the office environment.

In comparing the contaminant concentrations and comfort parameters to the standards, the most severe standards were used for the purpose of this study (see *Table 3* for a summary of the comfort and ventilation standards).

## Results & discussion

The measured values of chemical and gas contaminants are given in *Table 4*.

**Table 2. Health Regulations and Comfort Standards for Aircraft**

| Contaminant | Canadian Labor Code | Federal Aviation Administration | ASHRAE 62-1989 | Environmental Protection Agency | Exposure Guidelines/ Residential IAQ |
|---|---|---|---|---|---|
| Carbon dioxide (ppm) | 30,000<br>15 min.<br>5,000<br>8 hrs. | 30,000<br>continuous | 1,000<br>continuous | | < 3,500<br>continuous |
| Carbon monoxide (ppm) | 400<br>15 min.<br>50 ppm<br>8 hrs. | 50<br>continuous | | 35<br>1 hr.<br>9<br>8 hrs. | < 25<br>1 hr.<br>< 11<br>8 hrs. |
| Nitrogen dioxide (ppm) | 5<br>15 min.<br>3<br>8 hrs. | | | 0.05<br>1 yr. | 0.25<br>1 hr.<br>0.05<br>continuous |
| Formaldehyde ($\mu g/m^3$) | 3,000<br>15 min.<br>1,500<br>8 hrs. | | | | 120<br>continuous |
| Ozone ($\mu g/m^3$) | 600<br>15 min.<br>200<br>8 hrs. | 20<br>above 27,000 ft<br>3 hrs.<br>490<br>above 32,000 ft<br>continuous | | 235<br>1 hr. | < 240<br>1 hr. |
| Total dust ($\mu g/m^3$) | 10,000<br>8 hrs. | | | | 75<br>1 yr.<br>260<br>24 hrs. |

**Table 3. Ventilation and Comfort Standards**

| Parameters | ASHRAE 62-1989 | ASHRAE 55-1989 | ISO 7730 | EGRIAQ | Manufacturer Recomm. |
|---|---|---|---|---|---|
| Outdoor air supply rate cfm/person | 15 | | | | 10 |
| Temperature, °F (°C) | | 66-73 (19-23) winter<br>73-79 (23-26) summer | Vertical temp. diff. between ankles and head 3°<br>Floor temperature 66-79 (19-26) | | |
| Relative humidity (%) | | 20 (minimum) | | 30 | |

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The temperature and humidity results are shown in *Table 5*.

Ozone is prevalent at high altitude and during high latitude flights. Ozone enters the cabin from the outdoor supply air. The major acute and chronic health risk from inhalation exposure to this gas is irritation of the respiratory tract and lung tissue. Ozone was measured to be 26 percent of the limits allowed by the FAA health regulations and 2.3 percent of the EPA limit for 1 hour.

Carbon monoxide is a chemical asphyxiant, while nitrogen dioxide can cause irritation to the lung tissues. The major source of these contaminant gases is from the combustion of the engine fuel. Both gases were found to be substantially below the limits allowed by the regulations and comfort standards.

The sources of volatile organic compounds and formaldehyde can be from adhesives, lubricants, elastomers, sealing compounds, coatings, cleaning products, carpets and upholstery inside the aircraft cabin. The concentration of these contaminants is closely related to the amount of off-gassing of chemicals from these materials or from the volatization of the irresidual solvents.

Exposure to high concentrations of these chemicals affects the nervous system and respiratory tract. Volatile organic compounds were not detected, and formaldehyde compounds were found to be significantly less than the regulation health limits. Formaldehyde was measured at 20 percent of the level allowed by the most severe comfort standard.

The particulates are derived from the passengers' clothing and belongings, and from the activity on the runway during embarkment onto and disembarkment from the aircraft. The total particulate contents were measured to be substantially below regulation (1 percent) and 1.4 times that allowed by the most severe comfort standard.

An increase in ambient carbon dioxide in the aircraft results from the metabolism of the passengers released by exhalation. An increase in the number of passengers raises the carbon dioxide concentration in the aircraft.

Uncomfortable levels such as 50,000 ppm can cause headaches, dizziness and visual distortions. Symptoms relating to fatigue, headaches and stuffiness can be associated with levels between 500 and 3,200 ppm (FPA Committee 1987).

The measured concentrations did not exceed the FAA regulation limit of 30,000 ppm. However, in an aircraft carrying more than 74 passengers, the average carbon dioxide concentration was measured to exceed the ASHRAE *Standard 62-1989* limit of 1,000 ppm.

The general trend of carbon dioxide concentrations with respect to time (indicated by four flights on one excursion) is shown in *Figure 1*. The carbon dioxide concentration tends to peak at the beginning and at the end of each flight. This is taken as evidence that the fresh air supply level set by the aircrew is low during take-off and before landing.

The outdoor supply air contains outside air from the compressors in the engine and recirculated air from the cabin and storage areas. The quantity of outside air is regulated by a flow-control valve, controlled by the crew manually or automatically. Only one of the two air-packs can be functioning because of energy conservation (the pilot's decision is based on the number of passengers on-board).

From the compressors, the air passes through the heat exchangers where it is cooled or heated to the required comfort temperature. The supply air enters the cabin through the grilles situated on the sidewalls below the storage bins. The air is exhausted through the floor level grilles

**Table 4. Contaminants Measured In Aircraft Cabin**

| Contaminant | Mean | Mini. | Maxi. | Standard Deviation | Most Severe Health Regulation | Most Severe Comfort Standard |
|---|---|---|---|---|---|---|
| Carbon dioxide (ppm) | 719 | 330 | 2,170 | 233 | < 5,000 CLC | < 1,000 ASHRAE |
| Carbon monoxide (ppm) | 1.6 | 1 | 4 | 1.4 | < 50 FAA continuous CLC 3 hrs. | 9 8 hrs. EPA |
| Nitrogen dioxide (ppm) | < 0.1 | < 0.1 | < 0.1 | 0 | 3 8 hrs. CLC | 0.25 1 hr. EGRIAQ (regarding detectability) |
| Ozone ($\mu g/m^3$) | 5.3 | 5.3 | 5.3 | 0 | 20 continuous above 27,000 ft altitude (FAA) | 235 1 hr. EPA |
| Formaldehyde ($\mu g/m^3$) | 25 | 17 | 82 | 20 | 1,500 8 hrs. CLC | 120 continuous EGRIAQ |
| Volatile organic compounds ($\mu g/m^3$) | n.d. | n.d. | n.d. | n.d. | n.d. | n.d. |
| Total particulates ($\mu g/m^3$) | 105 | 25 | 200 | 68 | 10,000 8 hrs. CLC | 75 1 yr. EPA |

**Table 5. Temperature and Humidity Results**

| | Mean | Minimum | Maximum | Standard Deviation | Standard |
|---|---|---|---|---|---|
| Temperature, °F (°C) | 74 (23.4) | 56 (13.2) | 95 (35.1) | 1.6 | 67-73 (19.5-23) winter 73-79 (23-26) summer ASHRAE 55-1981 |
| Vertical temperature between ankles and head, °F (°C) | 2 (1.1) | 0 (0) | 4.3 (2.4) | 1.3 | 5.4 (3) ISO |
| Floor temperature, °F (°C) | 75 (24) | 61 (16) | 84 (29) | 2.3 | 66-79 (19-26) ISO |
| Humidity (%) | 18.5 | 4.6 | 48.5 | 3.8 | 30 EGRIAQ |

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that run alongside the wall and lead to the outflow valves. The amount of air exhausted is controlled as a function of the desired cabin pressure.

The results illustrated in *Figure 2* indicate that, generally, the quantity of outdoor air varies for each flight. When the number of passengers exceeds 34, the average quantity of outdoor air does not satisfy the manufacturers' standard of 10 cfm per person. When the number of passengers exceeds 13, the ASHRAE *Standard 62-1989* recommendation of 15 cfm per person is generally not met.

It was found that 18 out of the 33 flights evaluated did not comply with the aircraft manufacturer's recommendation (10 cfm) and 24 out of 33 flights did not satisfy the ASHRAE standard (15 cfm) for comfort.

In comparing *Figure 1* with *Figure 2*, it is evident that an increase in the carbon dioxide concentration does not necessarily correspond to a relatively low quantity of fresh air per person. Although it was noted that when the carbon dioxide concentration was above 1,000 ppm, the quantity of outdoor air per person generally averaged only 3 cfm per person.

Conversely, an acceptable concentration of carbon dioxide does not necessarily guarantee an acceptable quantity of outdoor air per person (as measured for flights 4, 6, 7, 8, 10, 12, 13, 16, 19, and 20 through 27). The results do not indicate a significant difference in the quantity of outdoor supply air or carbon dioxide concentrations between both sampling sites.

Temperature is controlled by the flight crew. The heat exchanger valve is used to establish the required cold temperature, whereas, when hot temperature is required, a zone reheating system exists to warm the air. The mean measured ambient air temperature of 73°F (23°C) is comfortable in relation to the recommended ASHRAE standard for the summer and winter seasons. The average vertical air temperature difference in the areas between the ankles and head is less than 5.4°F (3°C), and the average floor temperature is between the the recommended 66°F (19°C) and 78°F (26°C). Both are acceptable according to the ISO standards (see *Table 5* for the results).

The humidity in the aircraft depends on the respiratory and perspiratory vapor given off by the passengers and crew. No other sources are supplied, other than those from the food preparation and lavatory areas. Humidity is removed by mechanical water separators before entering the engine turbine and cabin air supply duct.

The relative humidity in the cabin varies inversely with the quantity of outdoor air. The results indicate low humidity, about 10 percent below the comfort recommendations (see *Table 5* for the results).

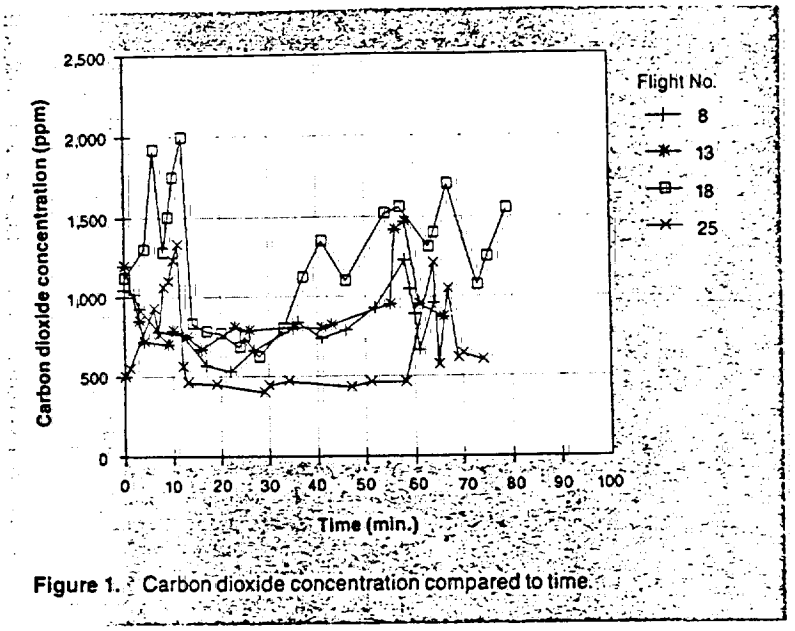


**Figure 1.** Carbon dioxide concentration compared to time.

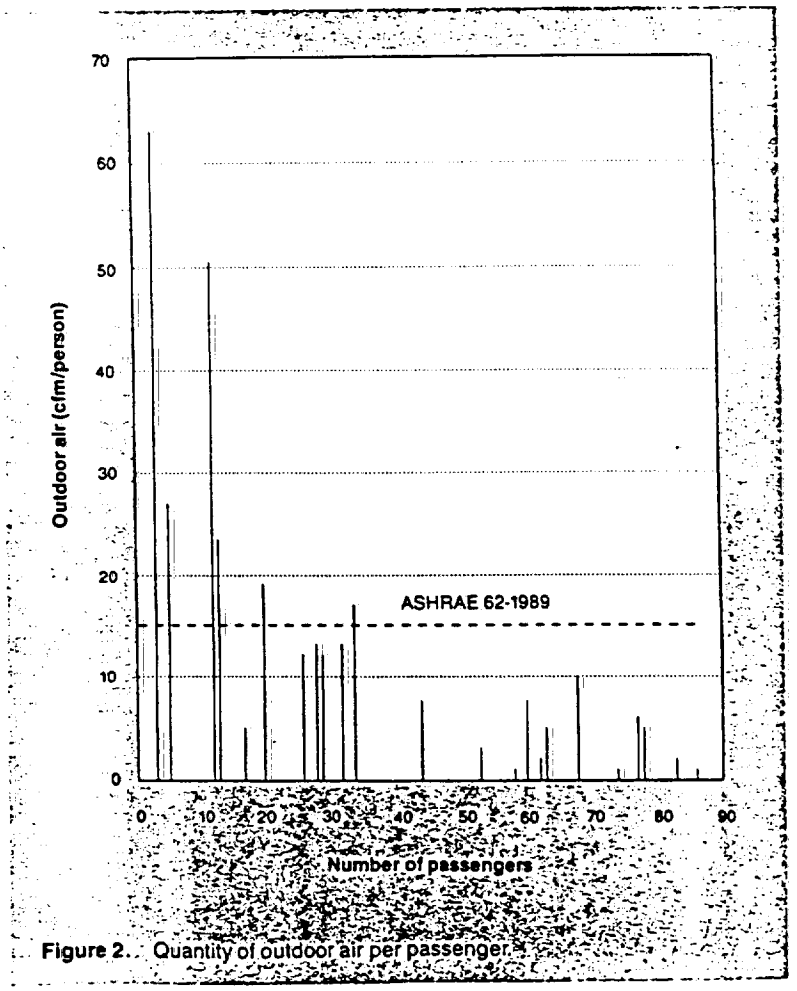


**Figure 2.** Quantity of outdoor air per passenger.

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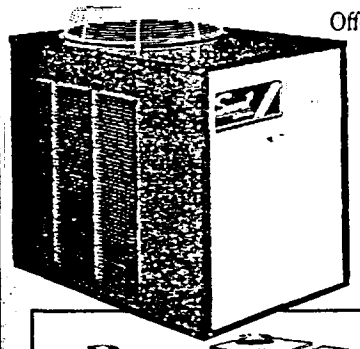
## Conclusion

The measured chemical contaminant levels were found to be within the regulations aimed at preventing a health risk. Only total particulate levels exceeded the comfort criteria even though smoking was not allowed on board.

The temperature results agree with respect to office building standards. The carbon dioxide concentrations were generally high when the number of passengers reached more than 68 percent of the aircraft occupancy. The outdoor air supply was generally below the ASHRAE recommended comfort level. Relative humidity could reach uncomfortable levels.

For improved comfort, the outdoor air supply should be increased on flights carrying more than 13 passengers. The desired increase in outdoor air supply should result in cabin concentrations of carbon dioxide below 1,000 ppm. Well-maintained air filters in the recirculating ventilation system would decrease the concentration of total particulates to a more comfortable level. Different options should be studied to attain an acceptable percentage of relative humidity. ■


# Gas Air Conditioning



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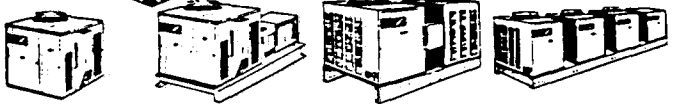

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## References


American Conference of Governmental Industrial Hygienists. 1986. *ACGIH Threshold Limit Values and Biological Exposure Indices for 1986-1987.* Cincinnati, Ohio.

ASHRAE. 1981. *Standard 55-1981, Thermal Environmental Conditions for Human Occupancy.* Atlanta, Georgia.

ASHRAE. 1989. *Standard 62-1989, Ventilation for Acceptable Indoor Air Quality.* Atlanta, Georgia.

Canadian Labour Code, Aviation Occupational Safety and Health Regulations. 1987. *Canada Gazette*, Pt. II, Vol. 121, No. 8, April.

Environmental Protection Agency. *National Primary and Secondary Ambient Air Quality Standards, Code of Federal Regulations*, Title 40, Part 50 (40 CFR 50).

Federal Aviation Administration. 1989. *Code of Federal Regulations: Aeronautics and Space.* Washington, D.C. Revised as of January 1, 1989.

Federal-Provincial Advisory Committee on Environmental and Occupational Health. 1987. *Exposure Guidelines for Residential Indoor Air Quality, Health and Welfare.* Ottawa, Canada. April.

Institut de recherche en sante et securite du travail (IRSST). 1988. *Guide d'echantillonnage des contaminants de l'air en milieu de travail.* Direction des Laboratoires. Montreal, Quebec.

International Standard Organization. 1984. *Standard 7730, Moderate Thermal Environments—Determination of the PMV and PPD Indices and Specification of the Conditions for Thermal Comfort.* Switzerland.

Manufacturer's ventilation recommendation. 1985. *Air Supply and Temperature Control.* Confidential report.

McGill University. *Formaldehyde Dosimeter Analytical Method.* School of Occupational Health Environmental Laboratories. Based on the NIOSH Method 3500, 1984, 3rd. ed.

National Institute of Occupational Health and Safety. 1977. *Manual of Analytical Methods*, 2nd ed. Publ. No. 77-157, 4 volumes. Cincinnati, Ohio. April.

National Research Council. 1986. *The Airliner Cabin Environment—Air Quality and Safety.* Report of the Committee on Airliner Cabin Air Quality, Board of Environmental Studies and Toxicology, Commission on Life Sciences. Washington, D.C.: National Academy Press.


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